

Compensatory Arm Reactions in Individuals with Parkinson's Disease

Tyler Weaver, BKin (Honours)

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Supervisor: Dr. Craig Tokuno, PhD

Faculty of Applied Health Sciences
Brock University
St. Catharines, ON.

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ABSTRACT

This study examined how perturbation-evoked compensatory arm reactions in individuals with Parkinson's disease (PD) are influenced by explicit verbal instruction. Ten individuals with PD and 15 older adults without PD responded to surface translations with or without specific instruction to reach for and grasp the handrail. Electromyographic (EMG) and kinematic recordings were taken from the reaching arm. Results showed that individuals with and without PD benefitted similarly from explicit instruction. Explicit instruction resulted in earlier ($p=0.005$) and larger ($p<0.001$) medial deltoid EMG responses in comparison to no specific instructions. Compensatory arm reactions also occurred with a higher peak medio-lateral wrist velocity ($p<0.001$) and higher peak shoulder abduction angular velocity ($p<0.001$) with explicit instruction. Explicit instruction positively influenced compensatory arm reactions in individuals with and without PD. Future research is needed to determine whether the benefits of instruction persist over time and translate to a loss of balance in real life.

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1.0 Review of Literature:

1.1 Balance Recovery in Humans:

1.1.1 Overview:

For humans to maintain an upright posture, their center of mass (COM) must remain positioned over the base of support (BOS) (Maki & McIlroy, 1997). The COM is defined as the point in which one's body mass is evenly distributed and when standing upright, is normally located slightly below one's belly button. The BOS is generally defined as the area created by one's body parts that are in contact with the ground. When standing upright, the BOS is the area underneath and between both of one's feet. The size of the BOS may be increased by increasing stance width or through the use of assistive devices such as canes or walkers. When one's COM moves beyond the limits of their BOS, they may experience a loss of balance and a fall may result.

To prevent a fall, two distinct categories of balance recovery strategies exist to bring the COM back over the BOS (Figure 1) (Maki & McIlroy, 1997).

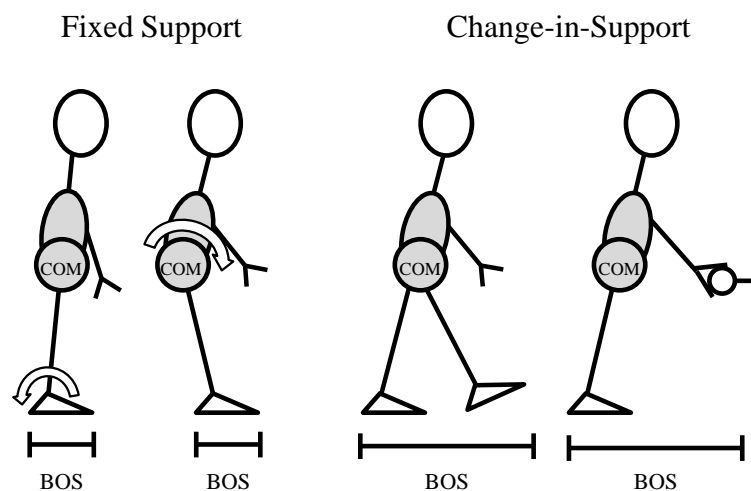


Figure 1: Balance recovery strategies. In change-in-support strategies the large increase in the size of the base of support (BOS) allows for a much larger range of center of mass motion to be accommodated. Figure adapted from Maki and McIlroy (2006).

A fixed support strategy commonly occurs in response to small or moderate perturbations to one's balance. These strategies do not change the size of the BOS but instead aim to stop movement of the COM by generating muscle torques and causing rotation about the ankle, knee and hip joints (Maki & McIlroy, 2006). However, when a larger perturbation is experienced, it may be necessary to rely upon a change-in-support strategy. A change-in-support strategy results in a large increase in the size of the BOS either through stepping or grasping an object (e.g., handrail) for support (Maki & McIlroy, 2006). Grasping a handrail is particularly beneficial because it helps to stabilize the COM over a fixed BOS (Maki & McIlroy, 1997) and anchor the body with respect to the environment (Maki & McIlroy, 2006). Consequently, such a strategy allows for a much larger range of COM motion to occur before it is brought to rest (Maki & McIlroy, 2006). Although the choice of balance recovery strategy is influenced by the size of the perturbation, an individual's balance capabilities will also determine whether a fixed or change-in-support strategy is selected. For example, older adults tend to rely more heavily on change-in-support strategies in order to recover balance. Video surveillance of a geriatric facility showed that stepping or grabbing nearby objects for support were quite common in response to a loss of balance (Holliday, Fernie, Gryfe, & Griggs, 1990). Of the 22 falls recorded, protective reaching occurred in 14 (64%) cases, with grasping observed in an additional 10% of cases.

1.1.2 Control of Compensatory Arm Reactions:

The high reliance on the arms for reaching with or without grasping a handrail, collectively referred to as compensatory arm reactions, has led researchers to examine

how these arm movements are neurally controlled. To evoke compensatory arm reactions in a laboratory setting, previous studies have used mechanical equipment to apply an external perturbation to the participant. The majority of these perturbations have been in the form of surface translations and rotational perturbations applied to the participant's BOS while standing upright (Allum, Carpenter, Honegger, Adkin, & Bloem, 2002; Bateni, Zecevic, McIlroy, & Maki, 2004; Ghafouri, McIlroy, & Maki, 2004; Mansfield & Maki, 2009; Mansfield, Peters, Liu, & Maki, 2010; McIlroy & Maki, 1995) or during locomotion (Dietz, Fouad, & Bastiaanse, 2001; Marigold, Bethune, & Patla, 2003; Misiaszek, 2003; Pijnappels, Kingma, Wezenberg, Reurink, & van Dieen, 2010; Roos, McGuigan, Kerwin, & Trewartha, 2008). Mechanical perturbations involving rapid chair tilts have also been applied to the seated participant (Gage, Zabjek, Hill, & McIlroy, 2007; Quant, Maki, Verrier, & McIlroy, 2001). In response to these perturbations, compensatory arm reactions have often been quantified based on electromyographic (EMG) recordings from the medial deltoid (Gage, et al., 2007; King, McKay, Cheng, & Maki, 2010; Mansfield & Maki, 2009), posterior deltoid (McIlroy & Maki, 1995), anterior deltoid (Ghafouri, et al., 2004), the biceps brachii (Bateni, et al., 2004; Gage, et al., 2007; Ghafouri, et al., 2004; Mansfield & Maki, 2009; Mansfield, et al., 2010; Quant, et al., 2001) and triceps brachii (Gage, et al., 2007). Kinematic measures from the wrist (Gage, et al., 2007; Ghafouri, et al., 2004), elbow (McIlroy & Maki, 1995) and shoulder (McIlroy & Maki, 1995) have also been analysed.

Results from these studies indicate that the EMG onset latency, or the time it takes for the upper arm muscles to activate following an unexpected loss of balance, is very rapid. In healthy young adults, upper arm muscle activity occurs less than 200 ms

following a loss of balance (Allum, et al., 2002; Bateni, et al., 2004; Gage, et al., 2007; Ghafouri, et al., 2004; Maki, Edmondstone, Perry, Heung, & Quant, 2001; Mansfield & Maki, 2009; Mansfield, et al., 2010; Marigold, et al., 2003; McIlroy & Maki, 1995; Misiaszek, 2003). Since the onset of upper arm muscle activation coincides temporally with the early activation of the ankle (Allum, et al., 2002; McIlroy & Maki, 1995; Misiaszek, 2003) and hip muscles (Allum, et al., 2002), a common central mechanism is suggested to be responsible for simultaneously activating the arm and leg muscles (Misiaszek, 2003).

Although the precise mechanism responsible for initiating compensatory arm reactions is still unknown, several possibilities exist. For example, some researchers have suggested that due to their very rapid initiation compensatory arm reactions in response to an external postural perturbation are stereotyped, startle responses (Hsiao & Robinovitch, 1998). However, since responses in the upper arm muscles are sensitive to the direction of the external postural perturbation (Allum, et al., 2002; McIlroy & Maki, 1995), startle reflexes alone cannot account for the initiation of the upper limbs when balance is perturbed. Instead, it is suggested that information received from the cutaneous afferents of the plantar surface of the feet may signal when standing balance is perturbed and lead to a rapid activation of the arms (Misiaszek, 2003). However, compensatory arm reactions are activated as quickly when participants are standing or sitting, when sensory information from the plantar surface of the feet or muscle spindle input from the ankles are not relied upon (Maki & McIlroy, 1997). Thus, additional sources of sensory information, such as the vestibular system, may also play an important role in the triggering of these arm reactions (Maki & McIlroy, 1997). The rapid initiation of the

arms may also be controlled by complex neural circuitry, in which primarily proprioceptive signals (e.g., muscle spindle input from the ankle) trigger specific pathways within the spinal cord (Marigold, et al., 2003). These specific proprio-spinal pathways may also receive cortical input to coordinate both the upper and lower limbs. The CNS can then fine tune these pathways as it adapts to repeated perturbations (Marigold, et al., 2003).

Although compensatory arm reactions occur in response to a loss of balance, previous research investigating the role of vision in compensatory arm reactions suggests that compensatory arm reactions may not be entirely controlled through reactive mechanisms. Ghafouri et al. (2004) found that the initial trajectory of the arm movement was functionally modulated depending on the direction of the external perturbation and the resulting direction in which the participant fell with respect to an invariantly located handrail. However, this arm movement was not dependent on whether vision was available or occluded at perturbation onset. Thus, the authors concluded that online visual input is not required to control compensatory arm reactions and instead, the CNS uses visual input stored prior to perturbation onset to pre-form a visual-spatial map. This information is then combined with online feedback from non-visual input (e.g., vestibular, proprioceptive) to fine-tune the motor response (Ghafouri, et al., 2004).

. Such pre-programming can be enhanced by providing information prior to a perturbation, in the form of invariantly located handrails (Bateni, et al., 2004; Ghafouri, et al., 2004; E. C. King, et al., 2010; Mansfield & Maki, 2009; Mansfield, et al., 2010; McIlroy & Maki, 1995) and explicit instruction to reach for and grasp the invariantly located handrails (Gage, et al., 2007; Ghafouri, et al., 2004; Mansfield & Maki, 2009;

Mansfield, et al., 2010; Quant, et al., 2001). The combination of having a handrail located in the same position throughout an experiment and explicitly instructing participants to reach for and grasp the handrail may encourage earlier and larger compensatory arm reactions by allowing individuals to incorporate this information to sculpt their compensatory arm reactions prior to losing their balance (Ghafouri, et al., 2004).

This possibility was confirmed in a recent study, whereby an invariant handrail location combined with explicit instruction to reach for and grasp the handrail, resulted in an earlier and larger upper arm EMG response in comparison to when the handrail location was unpredictable to the participant (Weaver, Hamilton, & Tokuno, 2012). Thus, the previously reported modulation and rapid initiation of compensatory arm reactions are likely a result of a certain level of response pre-programming, as explicit instruction to reach for and grasp the handrail allows participants to be more cognisant of using their arms to recover their balance by reaching for and grasping a handrail. Explicit instruction may also result in the participant becoming consciously aware of a handrail's location. Consequently, studies have recently investigated novel handrail cueing systems to elicit more effective compensatory arm reactions in both young and older adults. (Maki et al., 2008; Scovil et al., 2007). Such novel handrail cueing systems have included proximity triggered flashing green LEDS, and auditory prompts (e.g., "attention, use the handrail") in attempt to make participants more aware of the using the handrail to aid in their balance recovery (Maki, et al., 2008).

It is clear that compensatory arm reactions are an important part of balance recovery in humans. Yet, little is known about how these reactions are altered following different neuromuscular impairments, such as Parkinson's disease (PD). Many studies

have investigated how fixed support strategies or compensatory stepping responses are altered in individuals with PD, but have ignored the role of the arms during balance recovery (Dimitrova, Horak, & Nutt, 2004; Horak, Dimitrova, & Nutt, 2005; Horak, Nutt, & Nashner, 1992; King, St George, Carlson-Kuhta, Nutt, & Horak, 2010). This is worrisome because with the high rates of falls reported in individuals with PD (Bloem, Grimbergen, Cramer, Willemsen, & Zwinderman, 2001; Wood, Bilclough, Bowron, & Walker, 2002), all potential strategies for successful balance recovery, including compensatory arm reactions, must be investigated.

1.2 Parkinson's Disease:

1.2.1 Introduction:

Parkinsonism is a clinical syndrome manifested by part or all of the symptoms of resting tremor, rigidity, bradykinesia and postural instability (Caird, 1991; Hou & Lai, 2008). The most common type of parkinsonism is the disease that was first described by James Parkinson in 1817, Parkinson's disease. It is sometimes referred to as idiopathic parkinsonism, with the term idiopathic meaning unknown cause, or paralysis agitans, which when translated to English means "shaking palsy" (Duvoisin, 1984b). Parkinson's disease is a specific, chronic, progressive neurodegenerative disease that affects about 100,000 individuals, or 1 out of 345, in Canada (*Parkinson's disease: Social and economic impact.*, 2003).

Voluntary movement is normally initiated by the cerebral cortex and is subsequently regulated by complex feedback loops involving the thalamus, basal ganglia, cerebellum and the cerebral cortex itself (Hou & Lai, 2008) (Figure 2).

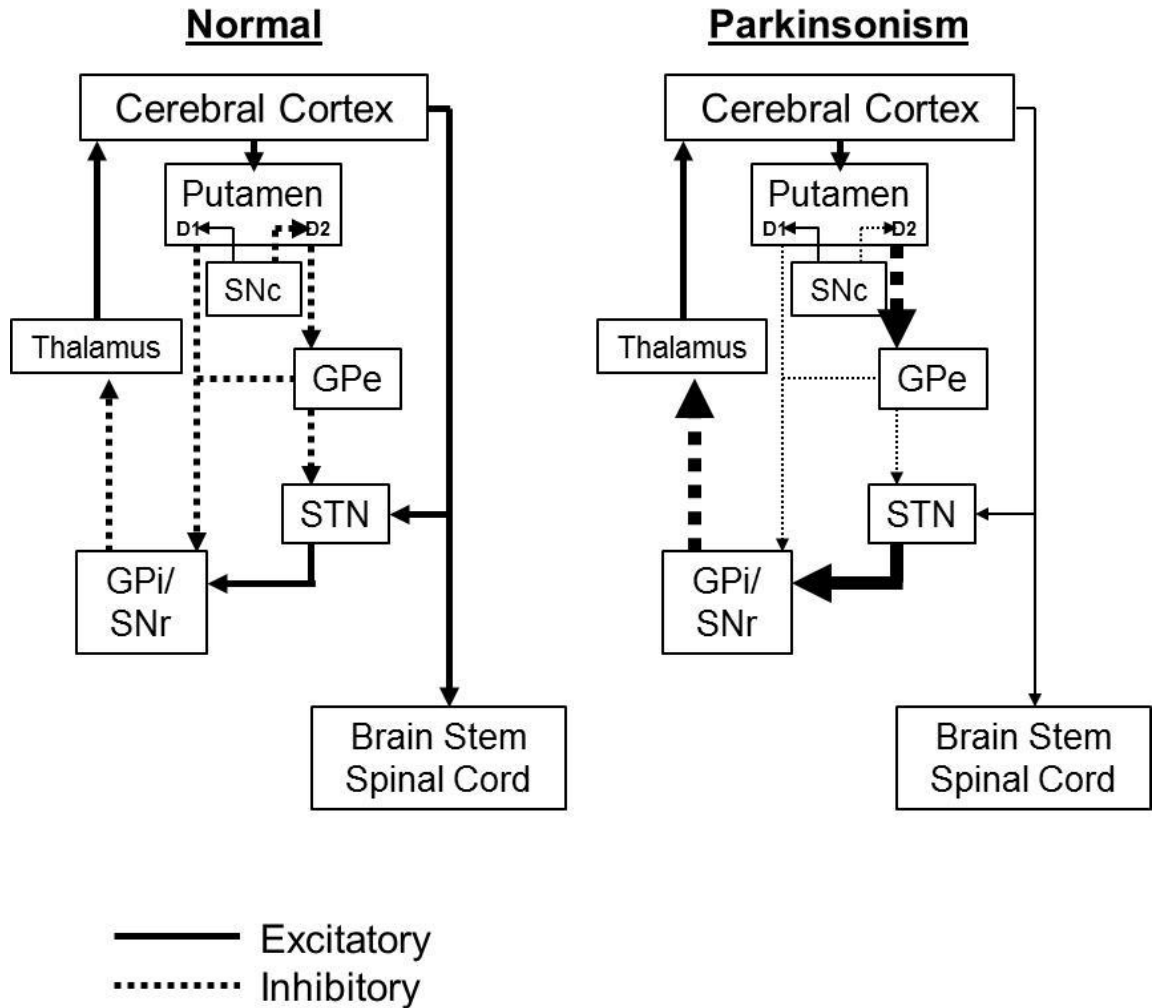


Figure 2: Basal ganglia circuitry in normal and parkinsonism patients. GPe and GPi refer to the globus pallidus external and internal respectively, while the SNc and SNr refer to the substantia nigra pars compacta and pars reticulata respectively.

The cerebral cortex, often times referred to as the command center, sends signals to the putamen and globus pallidus of the basal ganglia by the direct and indirect pathways (Hou & Lai, 2008). Neural signals from the direct pathway exit from the putamen to the globus pallidus internal, while neural signals from the indirect pathway exit from the putamen to the globus pallidus external, to the subthalamic nucleus and then reach the globus pallidus internal (Hou & Lai, 2008). In the nuclei of the putamen and the

globus pallidus internal and external, the primary neurotransmitter is gamma-aminobutyric acid (GABA). Within both the globus pallidus internal and external, GABA functions in an inhibitory role and therefore, both the globus pallidus internal and external have inhibitory effects on their targets. Dopamine in the substantia nigra regulates the putamen by using numerous dopamine receptors to enhance the inhibitory effect of GABA on the direct pathway and attenuate the effect of GABA on the indirect pathway (Hou & Lai, 2008). However in PD, dopaminergic neuronal loss in the substantia nigra results in a decreased level of dopamine, which results in disinhibition of the subthalamic nucleus and globus pallidus internal, and subsequently inhibits output from the thalamus to the cerebral cortex.

1.2.2 Motor Symptoms:

The decreased level of dopamine and the subsequent reduction in cerebral cortex activation that are associated with PD lead to various motor manifestations of parkinsonism, including resting tremor, bradykinesia and rigidity (Hou & Lai, 2008). The first motor symptom noticed in individuals with PD is usually resting tremor (Duvoisin, 1984a). As the name implies, resting tremor occurs when individuals are in the resting position and is commonly observed as a trembling or shaking of the hands at a frequency of between 4 to 6 Hz (Caird, 1991; Duvoisin, 1984a; Hou & Lai, 2008). However, the tremor can become more pronounced when the individual is under stress or with emotion (Caird, 1991; Duvoisin, 1984a; Hou & Lai, 2008). While the hands are the most common sites affected, the legs, chin, mouth and tongue may also be affected. The tremor typically begins on one side of the body and as the disease progresses, slowly involves the other

side of the body. However, it is usually the side with the initial symptoms that remains more severely affected.

Bradykinesia, or a slowness of movement, involves a prolongation of reaction time as well as a reduced velocity and acceleration once the movement begins (Hallett, Shahani, & Young, 1977). Patients may initially experience slowness or difficulty in performing simultaneous (Duvoisin, 1984a) or repetitive motor acts such as writing, but as time progresses, every motor act becomes laboured and less smooth (Hou & Lai, 2008). The patient's walking pattern also becomes slower and with shuffling steps (Caird, 1991; Duvoisin, 1984a; Hou & Lai, 2008).

Individuals with PD are also likely to experience rigidity, or an increase in muscle tone. Rigidity can cause a stooped posture, a forward shift of the body's COM as well as flexed limbs and reduced arm swing when walking (Caird, 1991; Hou & Lai, 2008).

1.2.3 Falls in Parkinson's Disease:

Due to bradykinesia and rigidity, individuals with PD are likely to experience postural instability. Postural instability may be a result of the loss of postural or righting reflexes (Caird, 1991) but more generally, postural instability also includes stooped posture and shuffling gait. As a result of involuntary propulsion during gait, individuals with PD are likely to fall forward. Additionally, they frequently experience freezing (e.g., a sudden inability and hesitancy in moving their legs) during gait initiation and turning, leading to an increased risk of falls during freezing (Hou & Lai, 2008). As a result of this postural instability, falling is a common problem for individuals with PD (Ashburn, Stack, Pickering, & Ward, 2001; Bloem, et al., 2001; Wood, et al., 2002).

In individuals with PD, falls are associated with reduced limits of stability, the use of small frequent steps during gait, and difficulty with dynamic balance when distracted (Ashburn, et al., 2001). Falling has also been shown to be strongly associated with disease severity (Ashburn, et al., 2001). The rate of falls in individuals with PD is between 50 and 70 percent (Ashburn, et al., 2001; Bloem, et al., 2001; Gray & Hildebrand, 2000; Wood, et al., 2002), with the risk of falling approximately twice that of community dwelling older adults (Wood, et al., 2002). Individuals with PD are also at a nine times greater risk of suffering recurrent falls in comparison to older adults without PD (Bloem, et al., 2001)..

Due to the increased risk of falls, individuals with PD also experience more fall-related injuries, including a higher rate of broken bones. Compared to older adults without PD, the majority of fractures in people with PD occur at the hip rather than the wrist (Grisso et al., 1991; Johnell, Melton, Atkinson, O'Fallon, & Kurland, 1992; Johnell & Sernbo, 1986; Pressley et al., 2003; Rison & Richardson, 2011). The high rates of hip instead of wrist fractures in individuals with PD is a concern because it suggests that they are unable to react and initiate compensatory arm reactions with sufficient speed to break their falls (Pressley, et al., 2003). Although this statistic implies that upper limb movements in response to a loss of balance may not be functional in individuals with PD, it is important to establish whether compensatory arm reactions are indeed altered in these individuals.

1.2.4 Compensatory Arm Reactions in Parkinson's Disease:

Despite the importance of compensatory arm reactions in balance recovery, only three research studies have investigated whether compensatory arm reactions are altered in individuals with PD. The first study examined how individuals with PD moved their arms in response to either a tilting bed or chair (Martin, 1965). Individuals with PD had arm reactions that were more limited in distribution and amplitude than individuals with no vestibular function, which the author attributed to impaired proprioception in individuals with PD (Martin, 1965). Although this study described the kinematics of compensatory arm reactions following PD, their findings do not provide detailed information of muscle activation patterns or specific kinematic parameters (e.g., arm angles or velocities). Thus, subsequent studies have obtained EMG recordings to better quantify the compensatory arm response in people with PD.

Carpenter, Allum, Honeggar, Adkin and Bloem (2004) examined how patients with PD respond to multi-directional postural perturbations when on (PD-ON) or off (PD-OFF) levodopa (L-dopa) medication. Participants were not specifically instructed on how to use their arms to help recover balance (Carpenter, et al., 2004). The PD-ON group demonstrated ~20 ms earlier medial deltoid responses compared to individuals without PD, but their arms did not move to a position where they could grab a handrail or cushion the force of a fall (Carpenter, et al., 2004). Additionally, while individuals with PD had initial arm pitch responses that were similar to the individuals without PD in pattern and timing, individuals with PD, both ON and OFF L-dopa, exhibited both a decreased velocity and peak displacement. In comparison to individuals without PD, both PD-ON and PD-OFF groups also exhibited decreased arm flexion in the pitch direction

and increased arm adduction (Carpenter, et al., 2004). There was a similar amount of reduction in arm movement observed between PD-ON and PD-OFF groups, indicating that while L-dopa results in earlier medial deltoid onset latencies compared to individuals without PD (~ 20 ms) it does not improve the ability of individuals with PD to use their arms to reach for a handrail in response to loss of balance (Carpenter, et al., 2004).

Visser et al. (2008) investigated the effects of subthalamic nucleus deep brain stimulation on axial and protective arm reactions in individuals with PD. Individuals with PD were tested with stimulation (STIM-ON) or without stimulation (STIM-OFF) but all individuals were on a supra-threshold (i.e., 150% of normal) dose of L-dopa prior to study commencement. The authors found that the amplitude of upper arm muscle activation was not statistically different between the STIM-OFF and the control (i.e., individuals without PD) groups. With respect to the arm elevation angle, no differences between STIM-OFF and individuals without PD, or STIM-ON and STIM-OFF patients were demonstrated. No direct comparison of arm muscle activity or arm elevation angle was made between the STIM-ON group and individuals without PD. Overall, the authors concluded that subthalamic nucleus stimulation had little influence on arm reactions and suggested that one reason for this observation was because individuals with PD took a supra-threshold L-dopa dose prior to the experiment, and that protective arm movements may be L-dopa responsive. However, this suggestion contrasts with the study by Carpenter et al. (2004), where PD-ON still did not exhibit biomechanically functional compensatory arm reactions (e.g., were not in a position to grasp a handrail due to decreased shoulder flexion and increased adduction). Thus, Carpenter et al. (2004) suggested that balance and postural responses in PD are not improved with L-dopa,

which is in line with previous studies which have suggested that L-dopa does not improve postural responses of the lower limbs (Bloem et al., 1996; Bloem, et al., 2001; Bloem, Munneke, Carpenter, & Allum, 2003; Jacobs & Horak, 2006).

1.2.5 Voluntary Arm Movements in Parkinson's Disease:

Due to the limited examination of how compensatory arm reactions are altered following PD, further insight may be gained by considering how voluntary arm movements are affected in individuals with PD. This is because both perturbation-evoked and voluntary arm movements share many similarities, including spatiotemporal muscle activity and kinematic measures, such that the two movement types are likely to involve similar control networks (Gage, et al., 2007).

Compared to healthy older and young adults, slower or bradykinetic movements have been observed in individuals with PD when performing rapid arm abduction movements (Baroni, Benvenuti, Fantini, Pantaleo, & Urbani, 1984), simple and complex arm movements (Agostino, Berardelli, Formica, Accornero, & Manfredi, 1992), arm point-to-point and reversal movements (Pfann et al., 2004), and reach-to-grasp movements (Rand, Lemay, Squire, Shimansky, & Stelmach, 2010; Rand, Smiley-Oyen, Shimansky, Bloedel, & Stelmach, 2006). Bradykinetic arm movements are also present regardless of whether individuals with PD have advance knowledge of the motor path (Curra et al., 1997). Individuals with PD were faster in executing the known motor sequence, indicating they were capable of using given information regarding the path of the required motor sequence to help execute their reaching movement. However, the

percentage of total movement time in the individuals with PD diminished less during the known motor sequence, in comparison to the individuals without PD (Curra, et al., 1997).

Bradykinetic voluntary arm movements by individuals with PD do not arise because of an inability to activate the agonist and antagonist muscles in the correct sequence. To the contrary, individuals with PD are able to activate both the agonist and antagonist muscles in the correct sequence, with normal antagonist inhibition (Hallett, et al., 1977) and with the appropriate anticipatory activity in postural muscles (Dick et al., 1986). However, their movements are performed with multiple cycles of EMG bursts of normal duration (Berardelli, Dick, Rothwell, Day, & Marsden, 1986; Hallett & Khoshbin, 1980; Hallett, et al., 1977). These multiple bursts of agonist and antagonist muscles have been shown to occur more frequently for movements of longer duration or amplitude (Hallett & Khoshbin, 1980). Berardelli et al. (1986) found that although patients with PD can produce large, long bursts of EMG activity, there is a failure to match the burst parameters to the size of the movement required. These individuals appear to underestimate what muscle activity is required for a particular movement due to a breakdown in the link between the perceptual appreciation of what is needed, and the delivery of the command to the motor cortex (Berardelli, et al., 1986). This perceptual-motor impairment during voluntary arm movements may be linked with the lack of functional arm reactions that occur in response to a loss of balance (Carpenter, et al., 2004). However, it is currently unknown whether both perturbation-evoked compensatory arm reactions and voluntary arm movements are affected to the same degree by PD.

1.2.6 Explicit Instruction: Verbal Instruction:

If perturbation-evoked and voluntary arm movements share similar control networks (Gage, et al., 2007), the suggested breakdown between perception and action during voluntary arm movements in individuals with PD (Berardelli, et al., 1986) may also affect perturbation-evoked compensatory arm reactions. Previously, a training program where individuals with PD were instructed to make large amplitude whole body movements (i.e., involving the head, arms, trunk and legs) was found to result in faster upper and lower limb movements 1 week post-training (Farley & Koshland, 2005). As such, perhaps compensatory arm reactions can be facilitated by explicitly instructing individuals with PD to initiate arm reactions in response to a loss of balance. It has been suggested that in individuals with PD, movement may be executed by using alternative motor pathways, bypassing the basal ganglia (Marsden & Obeso, 1994). For example, an intact cerebellar pathway may allow for individuals with PD to bypass the affected basal ganglia and allow these individuals to use visual input (i.e., transverse stripes on the floor during gait) to guide their movements (Glickstein & Stein, 1991). Thus, explicit instruction to reach for and grasp a handrail may help individuals with PD direct their gaze and attention to the handrail more frequently taking advantage of an intact visual-sensorimotor set (Jacobs & Horak, 2006) and bypassing the basal ganglia loop in the process.

The direction of one's gaze and attention to the handrail is important because some individuals do not fixate their gaze on a handrail in the environment prior to an unexpected loss of balance (King et al., 2011). Explicit instruction may direct a participant's gaze and attention toward the handrail at more frequent intervals prior to

perturbation onset, leading to a more frequent and successful use of the handrail for balance recovery. For example, it has been shown in young and older adults without PD, that when provided an auditory prompt to use a handrail, it led to an increase in the percentage of individuals who fixated on the handrail, both before and after a perturbation, as well as helped to reduce grasping errors (Maki, et al., 2008). In young adults, explicit instruction has also been shown to influence postural responses of the lower limbs, such that when instructed to keep their feet in place in response to a perturbation, the frequency of stepping tended to be lower in comparison to when no specific balance recovery response was required (McIlroy & Maki, 1993). Based on these findings, it seems logical to expect that explicit instruction provided prior to loss of balance may be beneficial in getting individuals with PD to reach for and grasp the handrail in response to a loss of balance. If improvements are observed in compensatory arm reactions when participants are explicitly instructed to reach for and grasp the handrail, then rehabilitation professionals may wish to incorporate explicit instruction into the training of these reactions in individuals with PD. Overall, this may allow individuals with PD to better use their arms when responding to a loss of balance, and potentially reduce the rates of hip fractures in these individuals.

2.0 Rationale, Purpose, Research Questions and Hypotheses:

2.1 Rationale:

It is currently suggested that compensatory arm reactions in individuals with PD are not functional. For example, Carpenter et al. (2004) reported that when individuals with PD were not explicitly instructed how to use their arms, they demonstrated greater

arm adduction in response to a loss of balance, in comparison to age-matched individuals without PD. This resulted in the arms not being in position to use the handrail to aid in balance recovery. However, this suggestion is based on the unconstrained study of these arm reactions (i.e., no specific instruction was provided to participants on how to use their arms for balance recovery). While it is important to study “natural” balance recovery responses, it remains unclear how individuals with PD would perform when a specific balance recovery response is desired (i.e., reaching for and grasping a handrail). Similar to how the frequency of reactive stepping in young adults tends to be reduced when participants are instructed keep their feet in place (McIlroy & Maki, 1993), it is of interest to determine if the frequency of handrail grasping can be influenced in individuals with PD when they are instructed to reach for and grasp the handrail in response to a loss of balance. Additionally, employing a method where an individual with PD is directed to use the handrail through explicit instruction, may be similar to how explicit visual cues improve compensatory steps in individuals with PD (Jacobs & Horak, 2006), possibly taking advantage of an intact visual-sensorimotor set. Explicit instruction may also encourage individuals with PD to fixate their gaze towards the handrail.

Therefore, this thesis addressed how the breakdown between perception (e.g., seeing the handrail) and action (e.g., reaching for and grasping the handrail) in individuals with PD affected their ability to execute compensatory arm reactions in a response to a loss of balance (Berardelli, et al., 1986). The goal of this thesis was to determine if explicit verbal instruction to reach for and grasp the handrail resulted in earlier and larger compensatory arm reactions in individuals with PD. More specifically,

participants were given the following instruction, “In response to the platform movement, recover your balance by reaching for and grasping the handrail using your left arm as fast as possible”. The responses from this condition were compared to an unconstrained condition where participants were given no specific instruction on how to recover their balance after the onset of a surface translation. Results from this thesis have potential future implications regarding the training of these rapid balance recovery reactions in individuals with PD and is an important first step in the possibility of training these balance recovery reactions. Future studies could then assess whether any potential improvements in these reactions transfer to more natural, unconstrained settings in which there is currently suggested to be a lack of functionality (Carpenter, et al., 2004). It seems possible that potential improvements would transfer, as implicit motor learning is thought to be preserved in individuals with PD (Abbruzzese, Trompetto, & Marinelli, 2009). Research in this area is important because individuals with PD are at a higher risk of fall-related injury, especially hip fractures (Pressley, et al., 2003).

2.2 Purpose:

The primary purpose of this study was to determine whether explicit instruction to reach for and grasp a handrail facilitates compensatory arm reactions in individuals with PD. The second purpose of this study was to determine whether deficits observed in perturbation-evoked compensatory arm reactions are different in comparison to voluntary arm movements (i.e., whether the observed deficits are specific to reactive or voluntary postural control).

2.3 Research Questions:

1) When examining compensatory arm reactions, do individuals with PD benefit more from explicit instruction (in comparison to no instruction) to reach for and grasp a handrail in response to a loss of balance, in comparison to older adults without PD?

2) Do compensatory arm reactions in response to an external postural perturbation differ from voluntary arm movements more or less in individuals with PD, in comparison to older adults without PD?

2.4 Hypotheses:

1) Individuals with PD will demonstrate an earlier EMG onset latency and larger EMG amplitude in the medial deltoid (MD) when explicitly instructed to reach for and grasp the handrail following a loss of balance. However, changes in EMG activity will be larger in comparison to older adults without PD.

2) Individuals with PD will demonstrate increased shoulder abduction, greater shoulder angular velocities, greater wrist displacement and greater peak wrist velocities when explicitly instructed compared to when not instructed to reach for and grasp the handrail. These improvements will lead to an earlier time-to-contact the handrail, compared to the no instruction condition. It is expected that the observed kinematic changes in individuals with PD will be larger in comparison to the older adults without PD, as individuals without PD are not affected by the motor symptoms (i.e., resting tremor, rigidity, bradykinesia and postural instability) associated with the disease. This will allow for individuals without PD to naturally exhibit faster and larger responses during the no instruction condition.

3) When compared to individuals without PD, perturbation-evoked compensatory arm reactions in individuals with PD will occur with smaller peak shoulder and wrist velocities and with a more limited shoulder abduction angle. Overall, PD will affect perturbation-evoked arm reactions to a greater degree in comparison to voluntary arm movements, despite improvements in perturbation-evoked compensatory arm reactions in individuals with PD when provided explicit instruction.

3.0 Methods:

3.1 Participants:

Ten individuals with PD (average \pm one standard deviation age of 74 ± 7 years; mass of 81 ± 18 kg; and height of 171 ± 7 cm) and 15 older adults without PD (average age of 69 ± 5 years; mass of 74 ± 21 kg; and height of 169 ± 11 cm) participated in the study (Table 1).

All participants were recruited from the Niagara Region through posters and word of mouth. Participants were excluded if they reported having any balance impairments not relating to PD, including disorders of the vestibular system, orthopaedic disease, musculoskeletal disorders or other neurological disorders that may have affected their balance. Participants who scored ≤ 24 on the Mini Mental State Examination or greater than 4 on the modified Hoehn and Yahr (HY) stages scale were also excluded from participating in the study. All participants provided their informed consent prior to participating in the study.

	Individuals with PD			Individuals without PD
	All (n=10)	“React Natural” First (n=5)	“Explicit Instruction” First (n=5)	All (n=15)
Sex	7 M, 3F	3 M, 2 F	4 M, 1 F	6 M, 9 F
Age (y)	74.2±7.3	74.2±6.0	74.2±9.4	69.1±5.4
Mass (kg)	80.9±18.3	83.4±25.0	78.5±10.5	74.4±20.5
Height (cm)	171±6.6	170.1±9.4	172±3.8	169.3±10.8
Time since diagnosis (y)	4.8±2.2	6±1.8	3.6±1.8	-----
MMSE (/30)	28±0.9	28.4±0.9	27.6±1.1	29±0.8
H & Y Score (/5)	2.0±0.6	2.0±0.7	2.0±0.7	-----
UPDRS Motor Score (/132)	34.2±10.1	33.7±11.9	34.7±9.6	-----
ABC Scale (/100)	81.2±13.6	83.4±7.4	78.9±18.8	90±8.1
Handedness	9 right; 1 left	4 right; 1 left	5 right; 0 left	14 right; 1 left
# of fallers in past year	4	1	3	2
TUG (s)	11.8±4.1	10.4±1.3	13.2±5.8	8.7±1.5

Table 1: Characteristics of participants with and without PD. Values represent the group mean ± one standard deviation. MMSE = Mini-Mental State Examination; H & Y = Hoehn & Yahr staging scale; UPDRS = Unified Parkinson’s Disease Rating Scale; ABC = Activities-specific Balance Confidence scale; TUG = Timed Up and Go test.

3.2 Questionnaires and Functional Assessments:

All participants first completed the Mini-Mental State Examination (Carpenter, et al., 2004; Folstein, Folstein, & McHugh, 1975) to allow for measurement of the participant’s cognitive status. Additionally, individuals with PD were assessed using the modified HY stages scale (Carpenter, et al., 2004), as well as Part III (motor section) of the Unified Parkinson’s Disease Rating Scale (UPDRS) (Visser, et al., 2008). The HY scale is widely used for describing broad categories of motor dysfunction in individuals with PD. It is capable of capturing typical patterns of progression of PD, both with and without dopaminergic therapy (Goetz et al., 2004). The modified HY scale incorporates

two additional levels, 1.5 and 2.5, which allow for axial involvement with unilateral signs and very mild postural impairment with recovery on the pull test (Goetz, et al., 2004). Although the scale is not intended to be a comprehensive exam, the HY scale remains an important descriptive, categorical scale.

The UPDRS captures multiple aspects of PD including motor impairment (Goetz et al., 2003). The UPDRS is unique because a teaching video-tape is available which standardizes the practical application of the scale and serves as an important asset to enhance inter-rater reliability (Goetz et al., 1995). Of all available clinical scales to assess PD motor impairment and disability, the UPDRS is currently the most widely used (Ramaker, Marinus, Stiggelbout, & Van Hilten, 2002), and has been shown to be a reliable and valid scale (Ramaker, et al., 2002).

Once it was determined that a participant (including the older adults without PD) met the inclusion criteria, they next completed the Activities-specific Balance Confidence (ABC) Scale (Powell & Myers, 1995) to assess their balance confidence as well as the Edinburgh Handedness Inventory (Oldfield, 1971) to determine individual handedness. Individuals with PD were also asked about how much time had elapsed since they were first diagnosed with PD, as well as which medications they had been prescribed, and were currently taking for their PD. The number of falls each participant had experienced in the last year was also recorded, where a fall was defined as, “an unexpected event in which the participant came to rest on the ground, floor, or lower level” (Lamb, Jorstad-Stein, Hauer, & Becker, 2005).

3.3 Experimental Setup:

EMG recordings were obtained from the MD of the left arm. To collect EMG recordings, the skin site of the MD was lightly shaved, abraded and cleansed with alcohol to limit the skin-electrode impedance. Disposable surface electrodes (10 mm diameter, 2 cm interelectrode distance, Kendall Meditrace 200, Mansfield, MA, USA) were then placed on the skin in a bipolar configuration over the muscle belly and oriented in the presumed direction of the underlying muscle fibres as determined by the origin and insertion of each muscle.

Three-dimensional kinematics were quantified using an Optotrak 3D Investigator system (Northern Digital Inc., Waterloo, Ontario). Clusters of four infra-red (IRED) markers were placed on the trunk, the left upper arm, the left forearm, and the two feet. Additional markers were also created with respect to the clusters of four IRED markers through digitization. These markers, referred to as imaginary markers, were digitized using a four-marker digitizing probe (Northern Digital Inc., Waterloo, Ontario) placed on the acromio-clavicular joint and the greater trochanter (bilateral); the anterior, medial, and poster deltoid of the left arm; the lateral and medial elbow of the left arm; the lateral and medial wrist of the left arm; the lateral and medial ankles of both feet; and the lateral and medial toes of both feet. These landmarks were digitized in order to provide position information regarding the proximal and distal ends of each segment.

3.4 Experimental Protocol:

All procedures were approved by the local university ethics review board prior to study commencement (file #11-152), in accordance with the Declaration of Helsinki. Of

the ten individuals with PD who completed the experimental protocol, eight were tested while on anti-Parkinson medication also referred to as the ON-state. This allowed for individuals with PD to be examined during their best clinical condition, about one hour after the intake of their anti-Parkinson medication. The remaining two individuals with PD had not yet been prescribed anti-Parkinson medication by their neurologist.

Participants stood on a 1.6 m x 0.9 m wooden platform that was affixed to the top of a computer-controlled motor driven platform. The participant's feet were kept shoulder width apart and their arms at their sides in a relaxed standing posture. The motor driven platform was a 4.3 m linear stage (H2W Technologies Inc., Valencia, CA, USA) that could translate in both the forward and backward directions. While standing on the platform, all participants wore a harness that was attached to a moveable overhead track to prevent them from falling to the ground. A spotter also stood on each side of the 1.6 m long wooden platform, within arm's reach of the participant, to provide an additional level of safety. In the event that a participant was unable to recover their balance on their own, the spotters prevented the participant from falling by providing external physical support. Throughout all experimental conditions, two vertical handrail supports were located on the left side of the participant, where a handrail measuring 1.98 m in length; and 0.04 m in diameter was placed. The handrail was located at 55 percent of each participant's total height and 25 percent of each participant's height away from the left hip (Weaver, et al., 2012) (Figure 3).

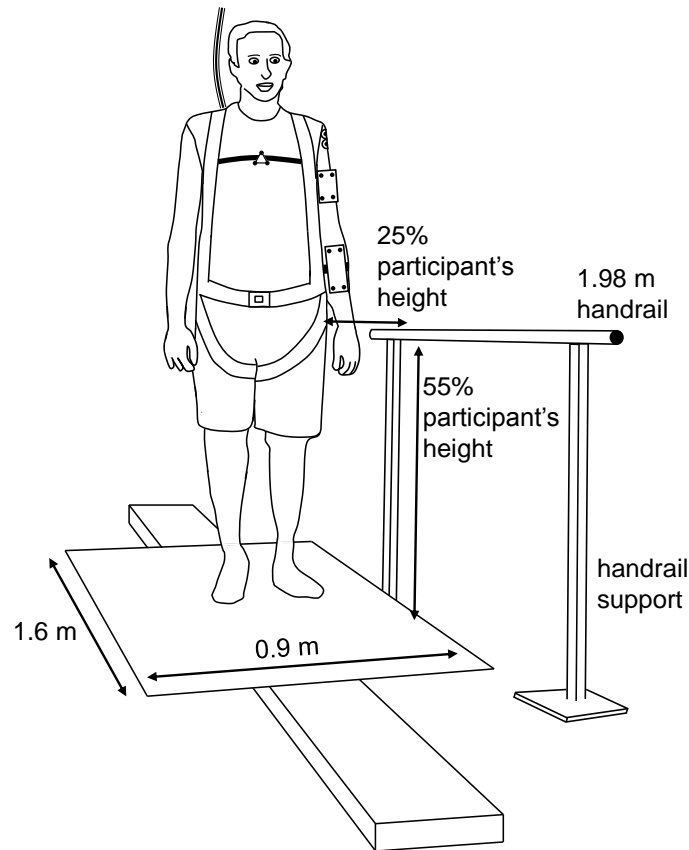


Figure 3 A diagram of the experimental setup depicting the participant standing on the 0.9 m x 1.6 m wooden platform, which was placed on top of a motor-driven 4.3 m linear stage. Vertical handrail supports were positioned such that a 1.98 m long handrail could be placed at a height of 55% of the participant's height and 25% of the participant's height away from the participant's left side. Although not illustrated, two spotters stood next to the platform for safety reasons.

All experimental conditions are detailed below. The two perturbation-evoked conditions were presented in two blocks of 15 trials. For the ten individuals with PD, half of the participants completed the “react natural” condition first, while the other half completed the “explicit instruction” condition first. For the 15 individuals without PD, the order of the two conditions was randomly assigned. Participants in both groups completed the two perturbation-evoked conditions first, followed always by the voluntary

condition. An attempt was made to match the two groups according to disease severity and age (Table 1).

3.4.1 Perturbation-evoked Trials:

This study administered 30 horizontal surface translations in the sagittal plane. Each surface translation was comprised of an initial peak acceleration of $1.2 \text{ m}\cdot\text{s}^{-2}$ for 450 ms, followed by a constant velocity of $0.4 \text{ m}\cdot\text{s}^{-1}$ for 300 ms, ending with a peak deceleration of $1.2 \text{ m}\cdot\text{s}^{-2}$ for 450 ms. Each surface translation displaced 0.30 m. This surface translation magnitude was chosen because it elicited compensatory arm reactions in the majority of participants (Weaver, et al., 2012).

Participants were tested in two perturbation-evoked experimental conditions: *react natural and explicit instruction*. For the react natural experimental condition, participants experienced 15 surface translations, with eight trials in the forward direction and seven trials in the backward direction. The order of translation direction was presented randomly. A greater number of forward trials were presented because individuals with PD exhibit their smallest stability margins in response to backward body sway (Horak, et al., 2005). In response to each surface translation, participants were instructed as follows: “In response to the platform movement you may do whatever comes natural to recover your balance”. This verbal instruction was presented only once, before the start of the first surface translation. No specific instruction was given with how they were to use their arms or legs in recovering their balance.

During the explicit instruction experimental condition participants were presented with 15 surface translations, with eight trials in the forward direction and seven trials in

the backward direction. The order of the platform direction was presented randomly. In response to each surface translation, participants were given the following instruction: “In response to the platform movement, recover your balance by reaching for and grasping the handrail using your left arm as fast as possible”. This instruction was presented verbally to the participant prior to the start of the first surface translation. If a participant did not follow the instruction, they were verbally reminded of the objective. Trials (4 out of 200) in which a participant did not follow the instruction (e.g., did not grasp the handrail when explicitly instructed) were removed from subsequent analysis.

3.4.2 Voluntary Arm Movement Trials:

Participants were tested in one voluntary experimental condition in order to enable comparisons in arm movement control between perturbation-evoked compensatory arm reactions and voluntary arm movements. For this condition, participants reacted to an auditory tone. The auditory tone had an intensity of 75 dB and lasted for 350 ms after onset. During this condition, participants were given the following instruction: “In response to the auditory tone, reach for and grasp the handrail using your left arm as fast as possible”. This instruction was presented verbally, and was given prior to the onset of the first auditory tone. A test auditory tone was also presented prior to the commencement of the condition in order to ensure that the participant could hear the tone clearly. Participants completed eight voluntary arm movement trials.

3.5 Data Collection and Analysis:

EMG recordings were amplified 350 times (MA-300, Motion Systems Inc., Baton Rouge, LA, USA) and analog-to-digital converted at a sampling rate of 2 kHz (micro1401, Cambridge Electronics Design, Cambridge, UK). The EMG recordings were then band-pass filtered offline between 60-600 Hz, rectified, and low pass filtered at 100 Hz. Kinematic data was collected at a sampling frequency of 100 Hz, and filtered offline using a Butterworth 4th order low-pass filter, with a cut off frequency of 6 Hz.

For each support surface translation trial, EMG onset latencies were determined as the time at which the rectified EMG signal exceeded a threshold of one standard deviation above the mean baseline for a period of at least 25 ms (Tokuno, Carpenter, Thorstensson, & Cresswell, 2006). Baseline mean and standard deviations for determining EMG onset latencies were calculated from the one second period before support surface translation or auditory tone onset. All EMG onset latencies were determined using a custom algorithm written within commercially available software (Spike2, Cambridge Electronic Design, Cambridge, UK) and later confirmed through visual inspection. Peak EMG activity, time-to-peak EMG activity and EMG offset latencies (i.e., the time when the EMG recording had an amplitude of less than 15% of its maximum signal) were also determined.

The area of the rectified EMG signal was calculated in order to determine the size of each muscle response after activation onset. The EMG area for each trial was calculated for two time intervals: 1) the 350 ms immediately preceding translation onset (i.e., background activity) and 2) 0-350 ms after muscle onset. A 350 ms window was chosen because MD EMG responses turned off within this time. To facilitate

comparisons between participants, EMG areas were normalized as a percentage of each participant's maximum voluntary contraction (MVC). MVC values were obtained while participants performed maximum isometric shoulder abduction (MD) against the external resistance of the experimenter. During these 2-s MVC contractions, the largest recorded EMG area over a 350 ms time window was considered to be 100% MVC. Unlike EMG area, peak EMG activity was not expressed as a percentage of MVC but instead was expressed in millivolts (mV).

The shoulder abduction angle was calculated as the angle between the upper arm and trunk segments because this angle corresponds closest with measured EMG activity from the MD (Visser, et al., 2008). The peak arm abduction angular velocity was also determined by differentiating the angular position data. The wrist position data was also differentiated in order to calculate the peak wrist velocity in the medio-lateral (ML) and vertical directions. From this data, the time-to-handrail contact was also determined, with movement completion defined as the point when the wrist velocity was less than five percent of peak wrist velocity in the ML direction (Ghafouri, et al., 2004).

3.6 Statistical Analysis:

Although participants experienced both forward and backward surface translations, only responses to forward directed translations were statistically analyzed. This was because individuals with PD have their smallest stability margins in response to backward body sway induced by a forward perturbation (Horak, et al., 2005).

Due to differences observed in the frequency of grasping (see section 4.0), two separate statistical analyses, one with all trials included and another with only the

grasping trials included, were conducted. By excluding the non-grasping trials for the react natural condition, changes in EMG or arm kinematics could be attributed to differences in instruction and not due to differences in the success of the reach and grasp response between instruction conditions. For each method, the effect of instruction on compensatory arm reactions was determined using a 2 x 2 x 2 mixed model analysis of variance (ANOVA), with group (individuals with vs. without PD) and order (“react natural” first vs. “explicit instruction” first) as randomized factors and instruction (react natural vs. explicit instruction) as a repeated factor. Instruction order was added as a between-subject factor in the ANOVA because the order of instruction presentation could confound the results through a learning effect due to experiencing repeated surface translation trials or through instruction carryover (McIlroy & Maki, 1993). For example, participants who performed the react natural condition as their second block of trials may have been more likely to grasp the handrail due to having previously performed the explicit instruction condition.

A 2 x 2 mixed model ANOVA was also conducted with group (individuals with vs. without PD) as a randomized factor and movement type (perturbation-evoked vs. voluntary) as a repeated factor, to determine how perturbation-evoked compensatory arm reactions differed from voluntary responses, when participants were provided explicit verbal instruction for both types of movement.

Non-parametric statistical tests were also used to compare the frequency of grasping between instruction conditions (Wilcoxon Signed Ranks test) and between both individuals with and without PD, and between instruction order (Mann-Whitney test). All significant interaction effects were explored through post-hoc paired sample t-tests.

Statistical significance for all tests was set at $p \leq 0.05$. All statistical calculations were performed using commercially available software (SPSS, Chicago, IL, USA). Data are presented as the mean \pm one standard error of the mean.

4.0 Results:

A summary of the resulting statistical effects observed during the two analyses can be found in Table 2.

Measure	All trials	Grasping trials only
MD EMG Background Activity	No effects	No effects
MD EMG Onset Latency	1; ($F_{1,21}=10.08$; $p=0.005$)	1; ($F_{1,13}=5.60$; $p=0.034$)
Time-to-peak EMG Activity	2; ($F_{1,20}=6.31$; $p=0.021$)	2; ($F_{1,13}=7.79$; $p=0.015$)
Peak MD EMG Activity (mV)	1; ($F_{1,20}=13.72$; $p=0.001$)	No effects
MD EMG area (0-350 ms)	1; ($F_{1,20}=41.09$; $p<0.001$) 5; ($F_{1,20}=4.00$; $p=0.059$)	3; ($F_{1,13}=7.51$; $p=0.017$) 5; ($F_{1,13}=3.33$; $p=0.091$)
Wrist ML Displacement	1; ($F_{1,19}=18.51$; $p<0.001$)	No effects
Peak Wrist ML Velocity	1; ($F_{1,19}=30.63$; $p<0.001$)	3; ($F_{1,11}=11.11$; $p=0.007$)
Peak Wrist Vertical Velocity	1; ($F_{1,19}=8.53$; $p=0.009$)	No effects
Peak Shoulder Abduction Angle	1; ($F_{1,20}=16.48$; $p=0.001$)	No effects
Peak Shoulder Angular Velocity	1; ($F_{1,20}=21.99$; $p<0.001$) 5; ($F_{1,20}=3.56$; $p=0.074$)	4; ($F_{1,12}=4.55$; $p=0.054$)

Table 2: Comparison of the statistical effects observed during the analysis of all trials vs. the analysis of only the grasping trials. 1=instruction main effect; 2= instruction x group interaction; 3=instruction x order interaction; 4=trend due to instruction; 5=trend due to group.

4.1 Influence of Instruction on Compensatory Arm Reactions for All Trials:

4.1.1 Frequency of Handrail Grasping:

The type of instruction given to the participant significantly influenced the frequency of handrail grasps ($Z=-2.96$; $p=0.003$). Grasping occurred in 126 out of 200 trials (63%) and 196 out of 200 trials (98%) during the react natural and explicit instruction conditions, respectively. The majority of the non-grasp trials during the react natural condition were a result of eight participants who grasped the handrail in only one or no trials. The frequency of handrail grasps was not different between individuals with and without PD, within each experimental condition (react natural: $Z=-0.15$; $p=0.878$; explicit instruction: $Z=-0.30$; $p=0.768$). Furthermore, the frequency of handrail grasps was not different based on instruction order, within each experimental condition (react natural: $Z=-1.08$; $p=0.279$; explicit instruction: $Z=-1.39$; $p=0.165$).

4.1.2 Electromyographic Data:

The background MD EMG area was not different between groups, instruction conditions or trial order. In response to a surface translation, the MD EMG onset latency was affected by an instruction main effect ($F_{1,21}=10.08$; $p=0.005$). For both individuals with and without PD, explicit instruction resulted in onset latencies that were 4% (6 ms) earlier compared to the react natural condition (139 ± 3 ms vs. 145 ± 3 ms).

The time-to-peak EMG activity in the MD was influenced by an instruction x group interaction effect ($F_{1,20}=6.31$; $p=0.021$). Post-hoc analyses revealed no difference in time-to-peak EMG activity between instruction conditions in individuals with PD ($t_9=0.65$; $p=0.530$). However, for the older adults without PD, the react natural condition

resulted in a 14 ± 4 ms earlier time-to-peak EMG activity compared to the explicit instruction condition ($t_{13} = -3.52$; $p = 0.004$).

Analysis of the peak MD EMG activity revealed an instruction main effect ($F_{1,20} = 13.72$; $p = 0.001$). Explicit instruction resulted in a 17% (0.13 ± 0.03 mV) increase in peak MD EMG activity compared to the react natural condition.

The MD EMG area 350 ms after muscle onset was influenced by an instruction main effect ($F_{1,20} = 41.09$; $p < 0.001$). Explicitly instructing participants to reach for and grasp the handrail resulted in a 32% increase in area compared to the react natural condition (33 ± 3 %MVC and 25 ± 3 %MVC, respectively) (Figure 4). The MD EMG area 350 ms after muscle onset was also influenced by a trend due to group ($F_{1,20} = 4.00$; $p = 0.059$), where individuals with PD tended to have a larger response than individuals without PD (36 ± 4 %MVC and 24 ± 2 %MVC, respectively).

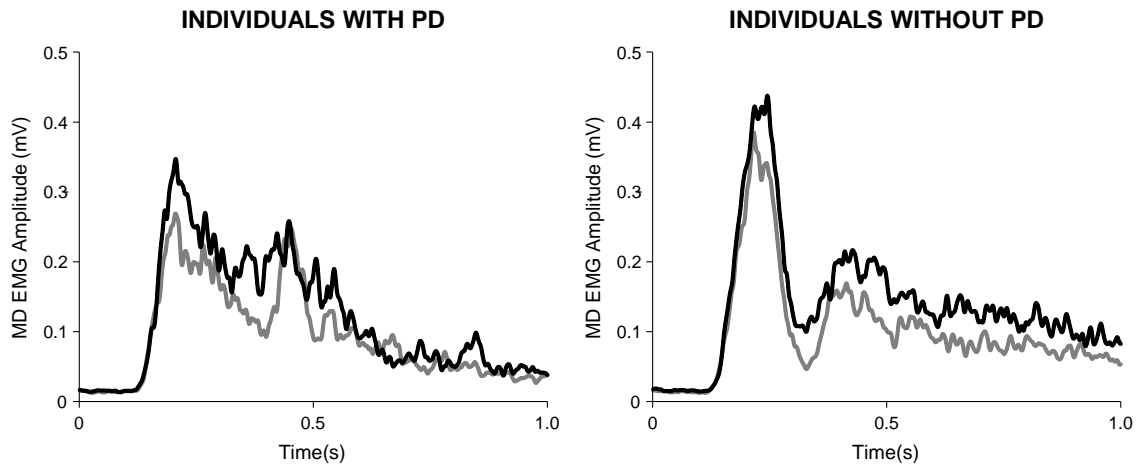


Figure 4: Point-by-point averaged medial deltoid (MD) electromyographic (EMG) traces obtained from all trials, for individuals with and without Parkinson's disease (PD). The black and gray traces represent the explicit instruction and the react natural conditions, respectively. All EMG data are time-referenced to the onset of the surface translation (time=0 s). Note the larger EMG amplitude during the explicit instruction compared to the react natural condition for both individuals with and without PD.

4.1.3 Kinematic Data:

Wrist Displacement and velocities:

The wrist ML displacement was influenced by an instruction main effect ($F_{1,19}=18.51$; $p<0.001$). A greater wrist ML displacement occurred during the explicit instruction (29.1 ± 0.8 cm) compared to the react natural condition (20.7 ± 1.6 cm). The peak wrist ML velocity also was influenced by an instruction main effect ($F_{1,19}=30.63$; $p<0.001$). Explicit instruction resulted in a 44% (52 ± 10 cm·s⁻¹) greater peak wrist velocity compared to the react natural condition (Figure 5).

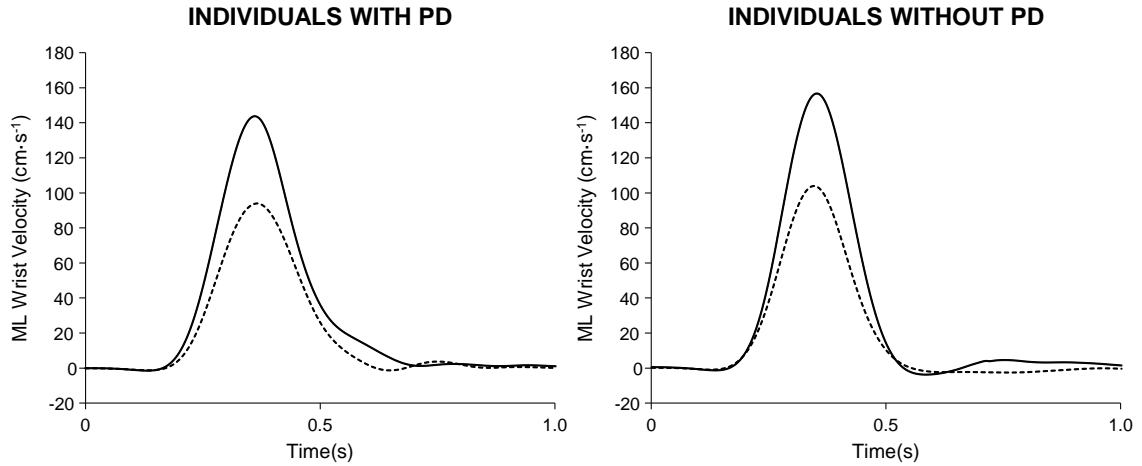


Figure 5: Point-by-point averaged medio-lateral (ML) wrist velocity traces obtained from all trials for individuals with and without Parkinson’s disease (PD). The solid and dashed lines represent the explicit instruction and the react natural conditions, respectively. All wrist velocity data are time-referenced to the onset of the surface translation (time = 0 s). A larger ML wrist velocity was observed when participants were provided explicit instruction compared to no specific instruction during the react natural condition.

Similar to the wrist ML velocity, the peak wrist vertical velocity was also influenced by an instruction main effect ($F_{1,19}=8.53$; $p=0.009$). A 22% (21 ± 7 cm·s⁻¹)

larger peak wrist velocity was found during the explicit instruction compared to the react natural condition.

Peak Shoulder Abduction Angle and Angular Velocity:

The peak shoulder abduction angle and angular velocity measures were influenced by an instruction main effect ($F_{1,20}=16.48$; $p=0.001$ and $F_{1,20}=21.99$; $p<0.001$, respectively). The shoulder abduction angle was greater during the explicit instruction ($43\pm1^\circ$) compared to the react natural condition ($35\pm2^\circ$). Similarly, explicit instruction resulted in a 29% greater angular velocity in comparison to the react natural condition ($190\pm10^\circ\cdot s^{-1}$ and $147\pm13^\circ\cdot s^{-1}$, respectively). The peak shoulder abduction angular velocity was also found to be influenced by a group trend ($F_{1,20}=3.56$; $p=0.074$), where individuals with PD tended to have slower shoulder movement in comparison to individuals without PD ($142\pm12^\circ\cdot s^{-1}$ vs. $184\pm11^\circ\cdot s^{-1}$, respectively) (Figure 6).

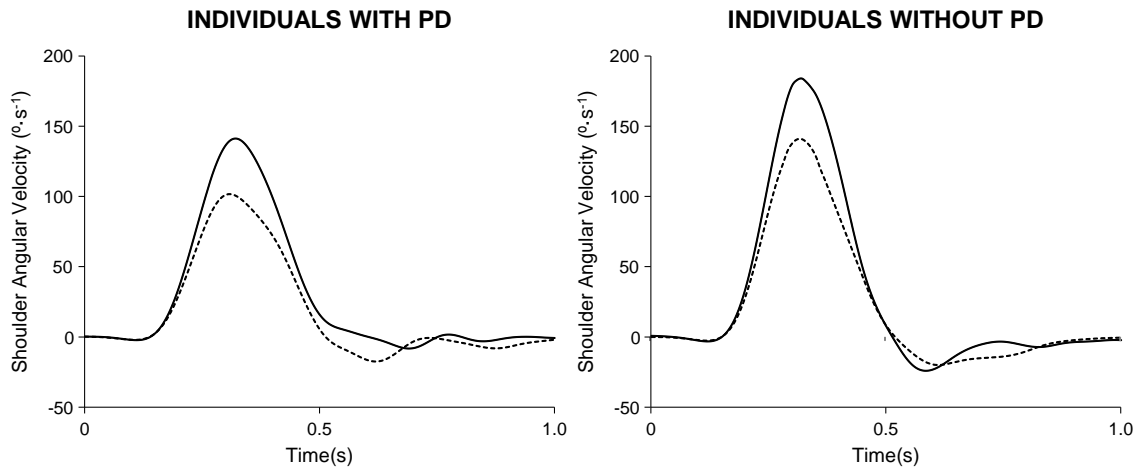


Figure 6: Point-by-point averaged shoulder abduction angular velocity traces obtained from all trials for individuals with and without Parkinson's disease (PD). The solid and dashed lines represent the explicit instruction and the react natural conditions, respectively. All angular velocity data are time-referenced to the onset of the surface translation (time = 0 s). Note the larger angular velocity during the explicit instruction compared to the react natural condition for both individuals with and without PD.

4.2 Influence of Instruction on Compensatory Arm Reactions: Grasping Trials Only:

4.2.1 Electromyographic Data:

When all trials were considered for analysis, an instruction main effect was found for the peak MD EMG activity; yet, when only the grasping trials were analyzed, no significant findings were observed for the peak MD EMG activity

During the analysis of all trials, the MD EMG area 350 ms after muscle onset was initially found to be influenced by an instruction main effect, with explicit instruction resulting in larger responses in comparison to the react natural condition. However, for the MD EMG area 350 ms after muscle onset, when only the grasping trials were analysed, an instruction x order interaction ($F_{1,13}=7.51$; $p=0.017$) was observed. Post-hoc analysis revealed that when the explicit instruction condition was completed first, explicit instruction resulted in larger responses in comparison to the react natural condition (36 ± 5 %MVC vs. 30 ± 5 %MVC; $t_{10}=-4.16$; $p=0.002$). In contrast, no difference was observed between the two instruction conditions when participants completed the react natural condition first (33 ± 3 %MVC vs. 32 ± 4 %MVC; $t_5=-0.37$; $p=0.728$) (Figure 7). Nonetheless, the group trend observed during the analysis of all trials persisted during the analysis of only the grasping trials.

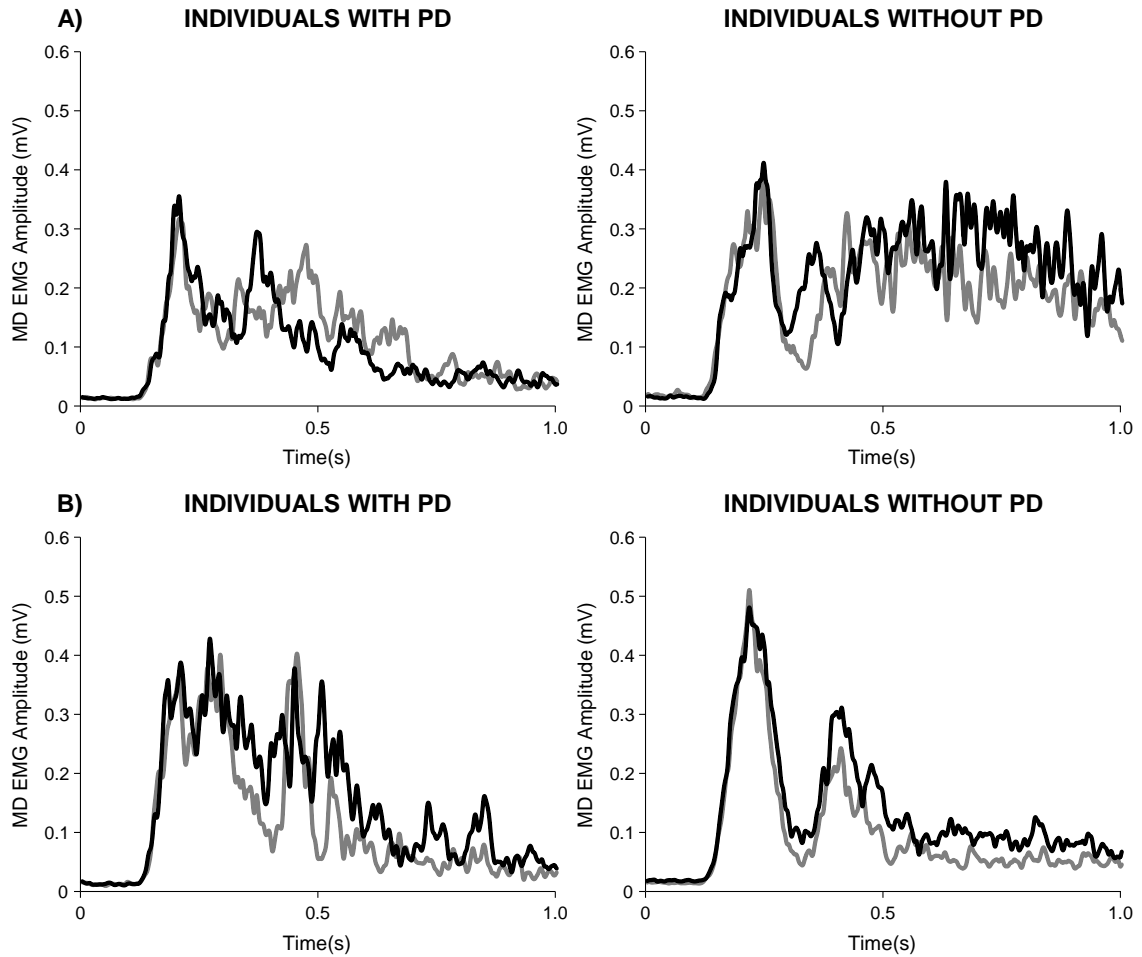


Figure 7: Point-by-point averaged medial deltoid (MD) electromyographic (EMG) traces obtained from only the grasping trials, for individuals with and without Parkinson's disease (PD). Participants who completed the react natural and explicit instruction conditions first are displayed in rows A and B, respectively. The black and gray traces represent the explicit instruction and the react natural conditions, respectively. All EMG data are time-referenced to the onset of the surface translation (time = 0 s).

4.2.2 Kinematic Data:

Wrist Displacement and velocities:

During the analysis of all trials, an instruction main effect was observed for the wrist ML displacement. Oppositely, analysis of the wrist ML displacement for only the

grasping trials revealed that the instruction main effect did not persist ($F_{1,11}=1.02$; $p=0.335$).

While the analysis of all trials revealed the peak wrist ML velocity to be influenced by an instruction main effect, analysis of the peak wrist ML velocity for only the grasping trials revealed an instruction x order interaction effect ($F_{1,11}=11.11$; $p=0.007$). When the react natural condition was performed first, peak ML wrist velocity was greater during the explicit instruction as opposed to the react natural trials (174 ± 11 vs. $144\pm 13 \text{ cm}\cdot\text{s}^{-1}$) ($t_4=4.95$; $p=0.008$). However, no difference was observed between the explicit instruction ($165\pm 12 \text{ cm}\cdot\text{s}^{-1}$) and the react natural ($159\pm 12 \text{ cm}\cdot\text{s}^{-1}$) trials when the explicit instruction condition was introduced first ($t_9=1.65$; $p=0.133$) (Figure 8). This suggests that participants may have learned from the explicit instruction to respond with higher peak ML wrist velocities during the subsequent react natural condition.

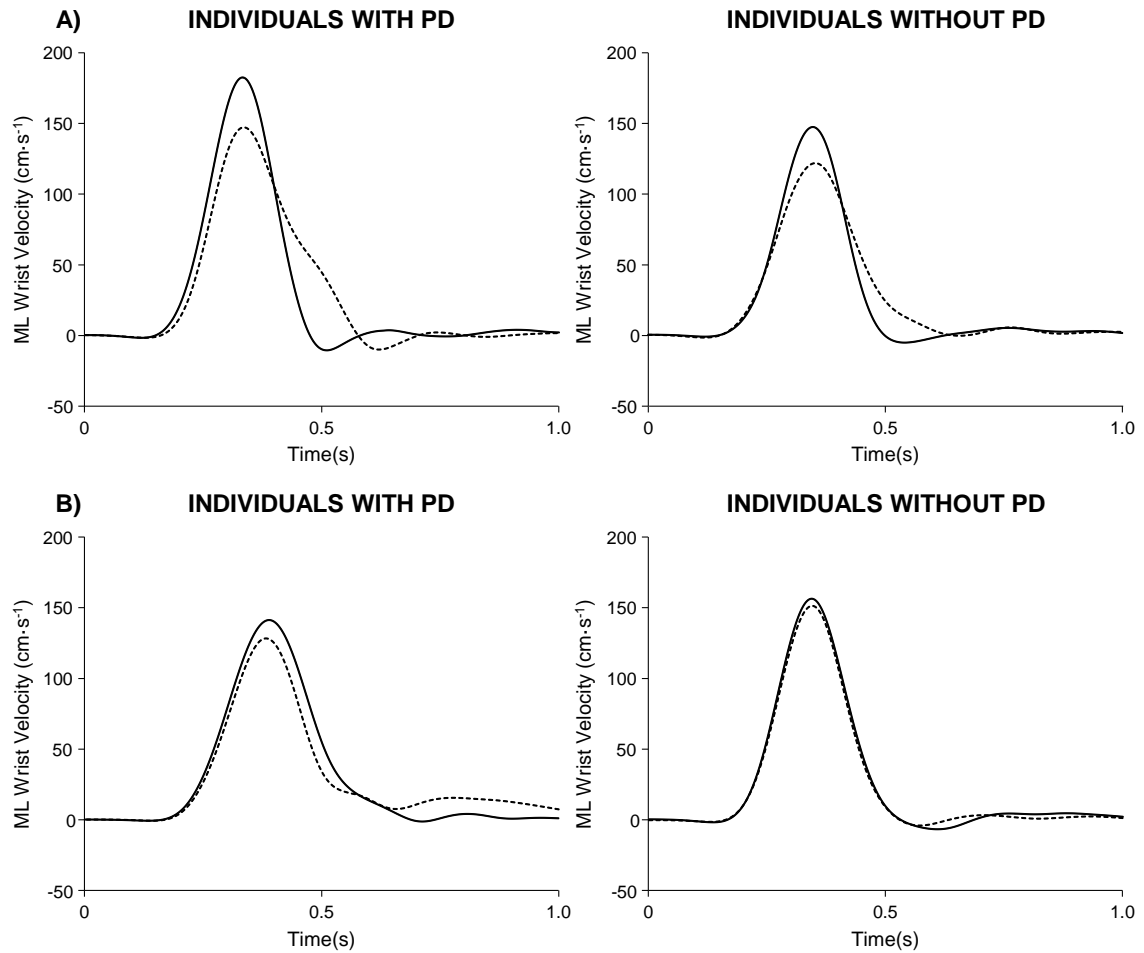


Figure 8: Point-by-point averaged medio-lateral (ML) wrist velocity traces obtained from only the grasping trials, for individuals with and without Parkinson's disease (PD). Participants who completed the react natural and explicit instruction conditions first are displayed in rows A and B, respectively. The solid and dashed lines represent the explicit instruction and react natural conditions, respectively. All wrist velocity data are time-referenced to the onset of the surface translation (time = 0 s).

While an instruction main effect was observed in the peak wrist vertical velocity when all trials were analyzed, this measure was not different between conditions when only the grasping trials were included for analysis.

Peak Shoulder Abduction Angle and Angular Velocity:

Whereas the peak shoulder abduction angle was influenced by an instruction main effect when all trials were analyzed, no significant effects were observed for this measure when only the grasping trials were analyzed.

Analysis of the peak shoulder abduction angular velocity during only the grasping trials showed the main effect of instruction, which was found to be significant during the analysis of all trials, to now be trending toward significance ($F_{1,12}=4.55$; $p=0.054$). The peak shoulder abduction angular velocity tended to be greater during the explicit instruction (196 ± 14 °·s⁻¹) compared to the react natural condition (185 ± 12 °·s⁻¹) (Figure 9). Additionally, the trend due to group which was observed during the analysis of all trials was no longer present during the analysis of only the grasping trials.

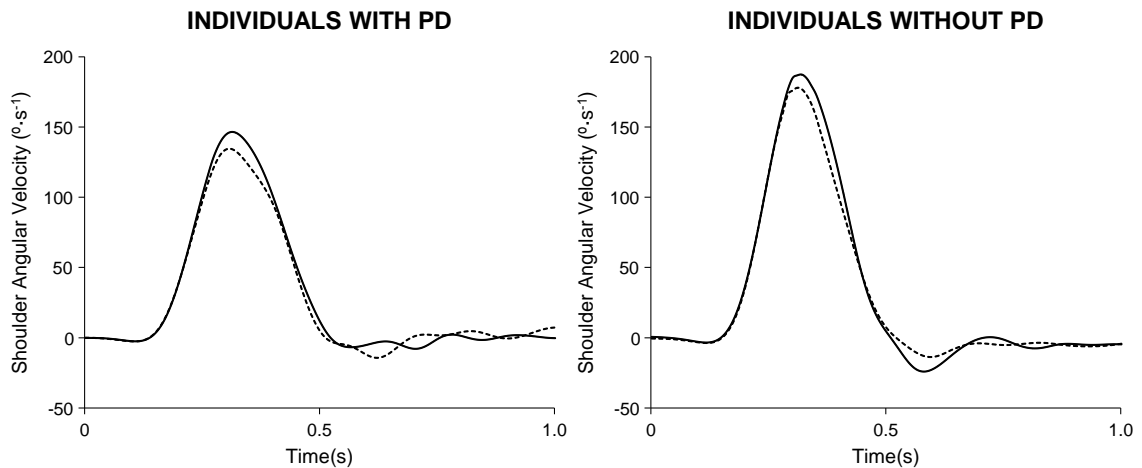


Figure 9: Point-by-point averaged shoulder abduction angular velocity traces obtained from only the grasping trials for individuals with and without PD. The solid and dashed lines represent the explicit instruction and the react natural conditions, respectively. All angular velocity data are time-referenced to the onset of the surface translation (time = 0 s).

Time-to-Handrail Contact:

The time-to-handrail contact was influenced by an instruction main effect ($F_{1,11}=8.37$; $p=0.015$). Participants were able to contact the handrail 49 ms earlier during the explicit instruction (521 ± 18 ms) compared to the react natural condition (570 ± 30 ms).

4.3 Perturbation-evoked vs. Voluntary Arm Movements:

4.3.1 Electromyographic Data:

The background MD EMG activity was influenced by a movement type main effect ($F_{1,22}=6.95$; $p=0.015$) and a group main effect ($F_{1,22}=5.20$; $p=0.033$). The movement type main effect was due to greater background EMG activity during the perturbation-evoked compared to the voluntary arm movement trials (2.0 ± 0.2 %MVC vs. 1.7 ± 0.2 %MVC). The group main effect was a result of higher background activity in individuals with compared to without PD (2.4 ± 0.3 %MVC vs. 1.4 ± 0.3 %MVC).

The MD EMG onset latencies were influenced by a movement type main effect ($F_{1,23}=31.09$; $p<0.001$), where the perturbation-evoked responses occurred 31% earlier than the voluntary responses (139 ± 3 vs. 182 ± 7 ms).

The time-to-peak MD EMG activity was not influenced by any main or interaction effects. However, a trend due to movement type was observed ($F_{1,22}=3.51$; $p=0.074$), where the perturbation-evoked responses tended to occur earlier in comparison to the voluntary movements (87 ± 4 ms vs 96 ± 4 ms). A group trend was also observed ($F_{1,22}=3.82$; $p=0.064$), where the individuals with PD tended to have an earlier time-to-peak MD EMG than individuals without PD (85 ± 5 ms vs 96 ± 3 ms).

Analysis of the peak MD EMG activity revealed a movement type main effect ($F_{1,22}=63.84$; $p<0.001$), where the peak EMG activity during the perturbation-evoked movements was 106% larger than the voluntary arm movements (0.85 ± 0.06 mV vs. 0.42 ± 0.03 mV).

Analysis of the 350 ms period post-muscle onset revealed that the MD EMG area was influenced by a movement type main effect ($F_{1,22}=77.65$; $p<0.001$) with perturbation-evoked responses being larger in comparison to the voluntary arm responses (33 ± 3 %MVC vs. 15 ± 1 %MVC). A group main effect ($F_{1,22}=5.32$; $p=0.031$) was also observed with individuals with PD having larger relative MD EMG responses in comparison to the older adults without PD (29 ± 3 %MVC vs. 20 ± 2 %MVC) (Figure 10).

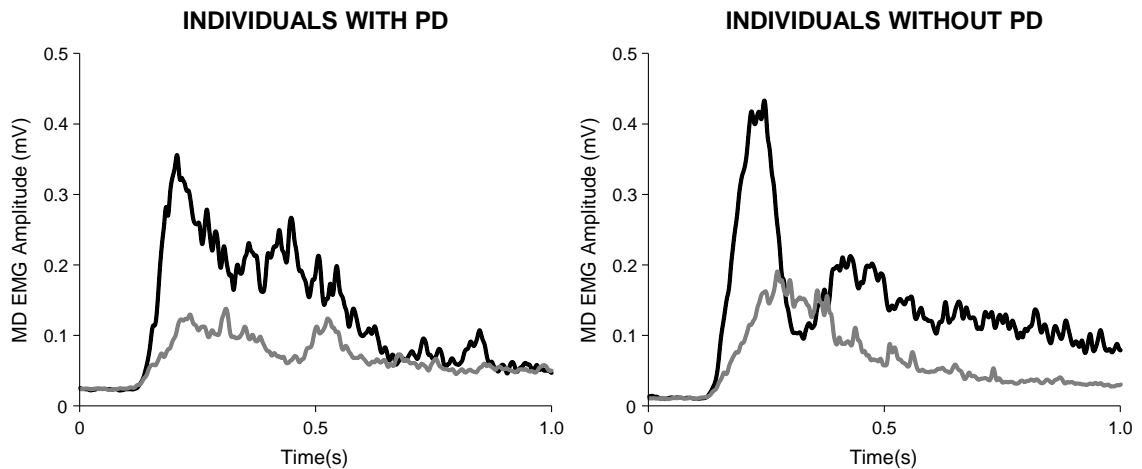


Figure 10: Point-by-point averaged medial deltoid (MD) electromyographic (EMG) traces for individuals with and without Parkinson’s disease (PD). The black and gray traces represent the perturbation-evoked and the voluntary reaching conditions, respectively. All EMG data are time-referenced to the onset of the surface translation or auditory tone (time = 0 s). Note the larger EMG activity during the perturbation-evoked compared to the voluntary reaching condition.

4.3.2 Kinematic Data:

Wrist Displacement and velocities:

The peak wrist ML displacement was influenced by a movement type main effect ($F_{1,21}=19.53$; $p<0.001$), with a larger displacement observed during the perturbation-evoked (29.1 ± 0.8 cm) compared to the voluntary arm movements (26.9 ± 1.0 cm).

Although the target handrail was in the same position for both reaching conditions, the difference in the peak wrist displacement may have been due to participants marginally overshooting the handrail during the perturbation-evoked condition where a rapid grasp of the handrail was important for balance recovery. However, it is debatable as to whether a difference of less than three centimeters is of functional significance.

Both the peak wrist ML and vertical velocities were influenced by movement type main effects ($F_{1,21}=26.96$; $p<0.001$; $F_{1,21}=83.53$; $p<0.001$). The peak wrist ML velocity was 24% larger during the perturbation-evoked movements (Figure 11), while the peak wrist vertical velocity was 44% larger during the perturbation-evoked compared to the voluntary arm movements.

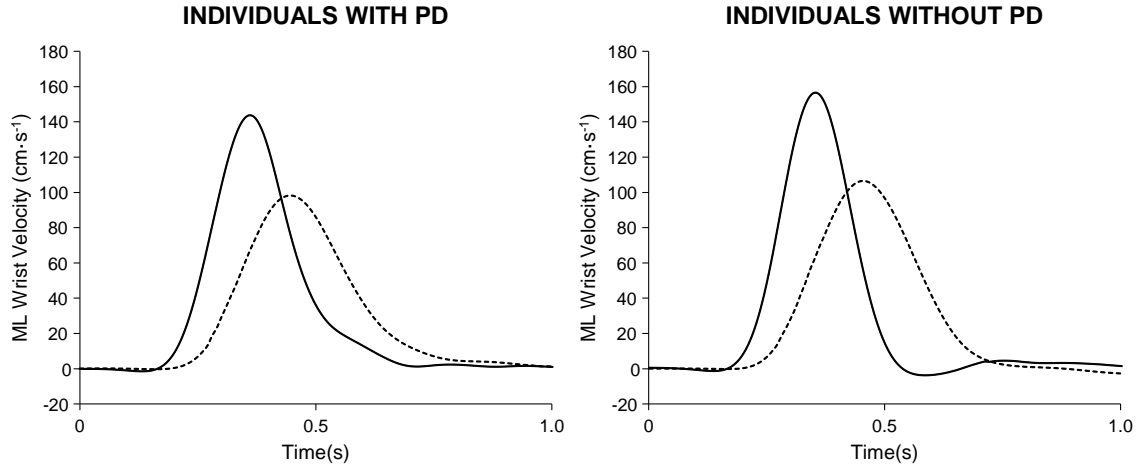


Figure 11: Point-by-point averaged medio-lateral (ML) wrist velocity traces for individuals with and without Parkinson’s disease (PD). The solid and dashed lines represent the perturbation-evoked and voluntary the reaching conditions, respectively. All wrist velocity data are time-referenced to the onset of the surface translation or auditory tone (time = 0 s). Note the larger wrist velocity during the perturbation-evoked compared to the voluntary arm condition.

Peak Shoulder Abduction Angle and Angular Velocity:

The peak shoulder abduction angle relative to the trunk was influenced by a movement type main effect ($F_{1,22}=26.43$; $p<0.001$), where the perturbation-evoked movements resulted in a larger angle of shoulder abduction in comparison to the voluntary arm movements ($43\pm1^\circ$ vs. $37\pm2^\circ$).

Analysis of the peak shoulder abduction angular velocity also revealed a movement type main effect ($F_{1,22}=29.75$; $p<0.001$) along with a group main effect ($F_{1,22}=9.91$; $p=0.005$). The movement type main effect arose because the peak angular velocity was greater during the perturbation-evoked compared to voluntary arm movements ($190\pm10^\circ\cdot s^{-1}$ vs. $137\pm8^\circ\cdot s^{-1}$). Further analysis of the group main effect revealed that the individuals with PD exhibited lower peak angular velocities of the

shoulder, in comparison to the individuals without PD (136 ± 11 $^{\circ} \cdot s^{-1}$ vs. 180 ± 9 $^{\circ} \cdot s^{-1}$) (Figure 12).

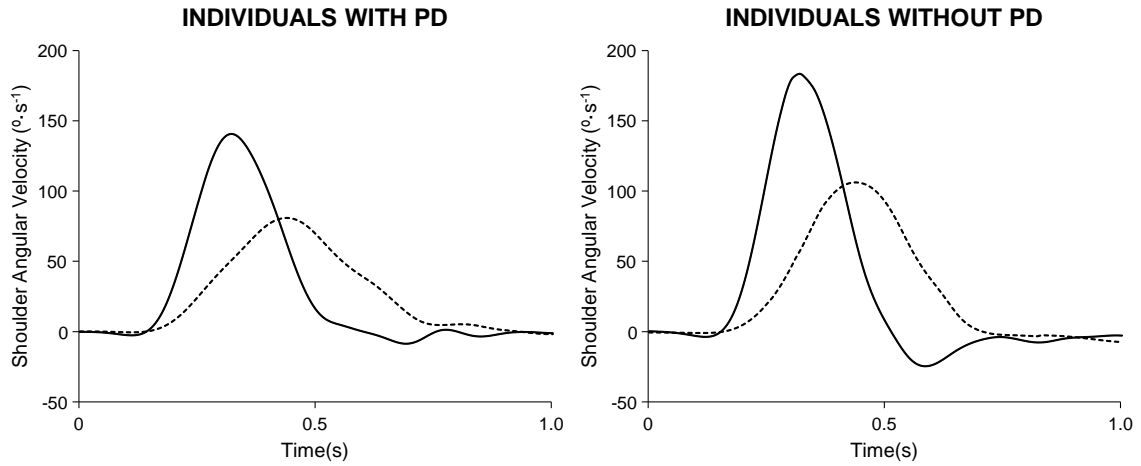


Figure 12: Point-by-point averaged shoulder abduction angular velocity traces for individuals with and without Parkinson’s disease (PD). The solid and dashed lines represent the perturbation-evoked and voluntary reaching conditions, respectively. All angular velocity data are time-referenced to the onset of the surface translation or auditory tone (time = 0 s). Note the larger angular velocity during the perturbation-evoked as compared to voluntary reaching condition and the smaller angular velocity in individuals with compared to without PD.

Time-to-Handrail Contact:

The time-to-handrail contact was influenced by a movement type main effect ($F_{1,21}=41.61$; $p<0.001$). Perturbation-evoked responses resulted in ~ 114 ms earlier time to contact the handrail compared to the voluntary arm movements (544 ± 18 ms vs. 658 ± 19 ms).

5.0 Discussion:

Previous research has suggested that compensatory arm reactions in individuals with PD do not provide any function in the process of balance recovery. Therefore, the

current study sought to determine whether explicit instruction to reach for and grasp a handrail facilitated compensatory arm reactions in individuals with PD. Overall, the results showed that explicit instruction positively influences compensatory arm reactions in individuals with PD, however this benefit was not limited to people with PD.

5.1 Influence of Instruction on Compensatory Arm Reactions:

By explicitly instructing participants to reach for and grasp the handrail prior to the loss of balance, earlier and larger MD EMG responses and a greater displacement and velocity of the shoulder and wrist segments were observed compared to the react natural condition. This along with the observation that the handrail was grasped more frequently during the explicit instruction condition shows the positive influence of explicit instruction on generating compensatory arm reactions. The facilitation of compensatory arm reactions with instruction is similar to how specific instruction to change specific gait variables (e.g., arm swing excursion, stride length, etc.) improves the walking patterns of individuals with PD (Behrman, Teitelbaum, & Cauraugh, 1998). The positive influence of instruction on compensatory arm reactions is also similar to how external cues impact gait in individuals with PD. For instance, both visual (e.g., brightly coloured parallel lines) and auditory (e.g., a metronome beat) cues have been shown to improve gait performance in individuals with PD (Morris, Iansek, Matyas, & Summers, 1996; Suteerawattananon, Morris, Etnyre, Jankovic, & Protas, 2004). This facilitatory effect resulting from the provision of instruction also resembles how instruction alters lower limb balance recovery strategies. For example, individuals with PD increase their reactive step length (Jacobs & Horak, 2006) and generate rapid steps when instructed to react to a

perturbation by stepping to a target (Maki & McIlroy, 2005). Similarly, instruction can both increase and decrease the frequency of reactive steps in individuals without PD (McIlroy & Maki, 1993; Weerdesteyn, Laing, & Robinovitch, 2008).

Contrary to our hypothesis, both individuals with and without PD grasped the handrail with a similar frequency, and the measured EMG and kinematic variables were not different between groups during both the react natural and explicit instruction conditions. Although some group main effects trended toward significance, the lack of significant between-group differences in initiating and activating compensatory arm reactions suggests that PD does not negatively influence compensatory arm reactions. This contrasts with the work of Carpenter et al. (2004) who suggested that individuals with PD exhibit compensatory arm reactions that are slower in the pitch direction, with reduced shoulder flexion and increased arm adduction in comparison to individuals without the disease. One potential factor that may explain the contrasting results is the number of handrails used in each study. Carpenter et al. (2004) employed three handrails, located to the front and both sides of the platform, and instructed participants to grasp the handrails only if needed. In contrast to multiple handrails surrounding the participant, the single handrail used in the current study meant participants were limited with respect to which arm they could use to reach for and grasp the handrail. The single handrail employed in the current study may have also acted as a visual cue, especially during the explicit instruction condition where participants were told to reach for and grasp the handrail. As such, the distinct, single handrail may have further directed the attention of the participants towards using it for balance recovery.

Another reason for the contrasting results between the current study and the work of Carpenter et al. (2004) may be explained by the constraints placed on the feet. In the study by Carpenter et al. (2004) the participant's feet were restrained, making it physically impossible for participants to take a step for balance recovery. In contrast, in the current study, no specific instruction or physical limitations were given to participants on how they were to use their legs or feet in recovering their balance. Although not restricting participant foot movement may have allowed for participants to prioritize stepping instead of reaching, this type of protocol better resembles what one would encounter in a real world situation. Specifically, individuals are more likely to respond to a loss of balance in daily life by reaching for a potential handrail and at the same time, taking one or more steps.

It is unclear why both groups benefitted equally from the provision of explicit instruction; however it may be that instruction to direct one's movement toward a specific target helps to reduce the contribution of sensory-motor pathways that are affected in PD. The provision of explicit instruction also likely allowed for an increased use of visual information about the handrail's location, which may have been beneficial to both individuals with and without PD. For instance, previous research has speculated that compensatory arm reactions are likely to be initiated through proprioceptive information (Marigold, et al., 2003); however proprioceptive-motor integration is suggested to be abnormal in individuals with PD when responding to a postural perturbation (Jacobs & Horak, 2006). Kinaesthesia (i.e., the perception of motion and direction of movements), which requires the processing of proprioceptive information, has also been shown to be impaired in individuals with PD during passive arm movements (Maschke, Gomez, Tuite,

& Konczak, 2003). Despite abnormal proprioceptive-motor integration, individuals with PD may be able to execute movement via alternative motor pathways which bypass the affected basal ganglia (Marsden & Obeso, 1994). For example, Glicktein and Stein (1991) suggested that an intact cerebellar pathway may allow for the use of visual input to guide movement, while the results of Kurata and Wise (1988) support the notion that the premotor cortex is involved in the generation of movement in response to a visual stimulus. In the current study, explicit instruction may have encouraged individuals with PD to direct their gaze to the handrail more frequently, allowing them to take advantage of visual input about the handrail's position. This would be similar to how visual input has been found to help increase compensatory step length in individuals with PD (Jacobs & Horak, 2006). Further, the use of visual information by individuals with PD to help control compensatory arm reactions is similar to how older adults without PD incorporate visual information about the handrail's location into the control of compensatory arm reactions when they are instructed to use a handrail (Cheng, McKay, King, & Maki, 2012). Given the parallel alterations in compensatory arm reactions with instruction between individuals with and without PD, the results suggest that PD does not influence the ability of older adults to incorporate stored visual information about a handrail's location into the control of compensatory arm reactions.

Another reason that may explain why individuals with and without PD benefitted similarly with instruction could be the mild level of disease severity demonstrated by the participants with PD, as the individuals who participated in this study demonstrated low to mild disease severity. The average duration of the disease was 4.8 years, with an average UPDRS motor score of 34.2 and average HY score of 2. Thus, the results of this

study may not be generalizable to people more severely affected by PD. However, it is also possible that based on the findings of Visser et al. (2008), disease severity alone has little effect on compensatory arm reactions. Clinical measures indicated that the participants in the study by Visser et al. (2008) were more affected by PD than the individuals in the current study, with a duration of 12.9 years, an average UPDRS motor score of 51.4 and average HY score of 3.3. Yet, similar to the current study, their participants were still able to produce compensatory arm reactions that were not different than the control group (i.e., individuals without PD). This lack of association between disease severity and the ability to produce compensatory arm reactions clearly warrants further investigation. Since clinical tests used to assess the severity of PD do not involve compensatory arm reactions, and only minimally assess reactive balance as a whole (i.e., retropulsion test), it may be difficult to draw conclusions on whether an individual with PD is able to rapidly reach for and grasp a handrail based on the results of clinical tests alone.

Finally, it is possible that the lack of group differences could be attributed to their medical state. Visser et al. (2008) reasoned that the lack of differences between groups may have arisen because their participants took 150% of their normal dose of L-dopa prior to study commencement. If compensatory arm reactions are L-dopa responsive as suggested, this would explain why this study did not find differences between PD and non-PD. However, the benefits of L-dopa for compensatory arm reactions are not definite, as Carpenter et al. (2004) observed small and abnormally directed arm movements regardless of whether a participant with PD was on or off their anti-Parkinson medication. As such, future research is needed to gain a better understanding of how

compensatory arm reactions are influenced by explicit instruction when individuals with PD are off their anti-Parkinson medication. This will help to determine whether the lack of group differences observed in the current study was a result of individuals with PD being tested when on their anti-Parkinson medication.

The lack of group differences can still be considered a positive outcome. The fact that the individuals without PD also benefitted from the provision of explicit verbal instruction supports previous research. For example, the use of an auditory cue to prompt handrail grasping is beneficial to both young and older adults in increasing the percentage of participants who fixated on the handrail both before and after a perturbation, as well as reducing the frequency of grasping errors (Maki, et al., 2008). Previous research also suggests that when provided with an auditory cue directing one's attention to a handrail, both older and younger adults are more likely to grasp the handrail, in comparison to when no cue is provided (Scovil, et al., 2007). Thus the current study further contributes to existing research suggesting that compensatory arm reactions in older adults without PD can be improved through external cueing. Employing techniques such as external cueing to improve compensatory arm reactions, and ultimately reducing the frequency and severity of falls, should be especially beneficial.

5.2 Influence of Instruction on Compensatory Arm Reactions: Grasping Trials Only:

It was important to separately analyze only the grasping trials in order to determine if instruction led to a change in the speed or muscle activity of compensatory arm reactions when the end result (i.e., handrail grasp) was the same. With this analysis, it was evident that some of the main effects of instruction did not persist. These measures

included the peak MD EMG activity, the peak wrist vertical velocity as well as the wrist ML displacement and peak shoulder abduction angle. It is not surprising that the two kinematic parameters were no longer different between instruction conditions because the target handrail which participants grasped was in the same location throughout all trials. Of the effects that continued to be significant when only the grasping trials were analyzed, the most important finding was that explicit instruction resulted in a 49 ms earlier time to handrail contact in comparison to the react natural condition. However, while a 49 ms earlier time to handrail contact is a positive result, it appears as though the primary benefit of explicit instruction to reach for and grasp the handrail is that it causes participants to grasp the handrail more frequently, as many of the outcome variables were not different when the handrail was successfully grasped.

While the results of the current study suggest that there is a positive effect of instruction on compensatory arm reactions in individuals with PD, this type of protocol is only appropriate if the adaptations are retained over time and transferable to other balance tasks and everyday activities. There is potential for improvements to be retained in individuals with PD, as previous research has shown that the benefits of a repetitive compensatory step training program, where individuals with PD were instructed to make large compensatory steps, persisted for two months without additional training (Jobges et al., 2004). In the current study, when only the grasping trials were analyzed, some short term benefits due to instruction were observed. Specifically, for the peak wrist ML velocity, an interaction effect showed that when individuals completed the explicit instruction condition first, the facilitated arm responses from the explicit instruction trials carried over onto the subsequent react natural trials. This suggests that participants

learned the importance of using their arms for balance recovery and maintained their wrist velocity during the subsequent unconstrained responses. This effect is similar to the influence of instruction order on feet in place responses (McIlroy & Maki, 1993). Specifically, when responding to a postural perturbation, individuals without PD were shown to be less likely to step when presented with an unconstrained task because of prior experience (i.e., carryover) with the instruction to keep their feet in place (McIlroy & Maki, 1993). The potential learning effect observed in the current study is also similar to the learning that occurs when individuals with PD repeatedly perform voluntary upper extremity movements (Felix et al., 2012). Since the current study was not designed to assess learning in more detail, future work should examine whether the effect of instruction on compensatory arm reactions in individuals with PD would persist over a longer period of time.

5.3 Perturbation-evoked vs. Voluntary Arm Movements:

The second purpose of this thesis was to compare perturbation-evoked to voluntary arm movements in individuals with and without PD. Statistical analyses revealed a movement type main effect for the majority of the measured variables. The differences in EMG onset latency and wrist velocity that were observed between the two arm movement types are in line with previous work. Gage et al., (2007) found that despite similarities in spatiotemporal muscle activity and kinematic measures, perturbation-evoked arm movements occur earlier and are executed faster in comparison to voluntarily initiated ones (Gage, et al., 2007; Weaver, et al., 2012). However, a main effect of movement type was also observed for the background MD EMG activity but

inspection of the numbers revealed an average difference between the two conditions of only 0.3 %MVC. Such a difference was unlikely to have been large enough to result in the movement type main effects observed in the EMG and kinematic variables post perturbation/auditory tone onset.

During the analysis of the voluntary arm movements, it was observed that the peak wrist ML displacement was greater during the perturbation-evoked compared to the voluntary arm movements. This is somewhat surprising, as the target handrail was in the same position throughout the duration of the study. However, the perturbation-evoked arm movements may have resulted in a larger wrist ML displacement because of the resulting postural instability caused by the surface translation. Participants may have overshot the handrail due to a need to move their arm laterally and grasp the handrail as fast as possible. This possibility would support previous studies that have found perturbation-evoked arm movements in older adults without PD to be more variable (Cheng, et al., 2012) and involve more undershooting and overshooting of the target handrail (Mansfield, et al., 2010) in comparison to young adults. The peak angle of shoulder abduction was also greater during the perturbation-evoked arm movements, which may have been a result of perturbation induced trunk movement, resulting in the observation of a larger angle between the trunk and upper arm, in comparison to the more stationary voluntary arm movements.

With respect to the individuals with PD, it was hypothesized that the presence of PD would have a greater influence on the perturbation-evoked compared to the voluntary arm movements. This was based on the lack of functional arm movement demonstrated by individuals with PD when responding to a loss of balance (Carpenter et al. (2004).

However, no interactions between movement type and group were observed. This suggests that perturbation-evoked and voluntary arm movements are not differentially affected by PD. There were also few significant group main effects. One of the group differences observed was with respect to peak shoulder abduction angular velocity, where individuals with PD exhibited lower peak shoulder abduction angular velocities across both types of movement compared to individuals without PD. It is not known why a similar group main effect was not observed during the analysis of only the perturbation-evoked trials. However, it may have been that there was greater variability in the react natural trials, thus preventing a group main effect from being observed. Oppositely, it may have been that there was a large difference within the voluntary trials, thus driving the observed group main effect during the comparison of the perturbation-evoked to voluntary arm movements. Nonetheless, the observation of bradykinetic movements by individuals with PD is not surprising, as this have been previously observed during both voluntary and perturbation-evoked arm movements (Agostino, et al., 1992; Baroni, et al., 1984; Carpenter, et al., 2004). It is also possible that the slower shoulder movements in individuals with PD may have been partially due to fatigue. While all participants completed the same experimental conditions and number of trials, perhaps the individuals with PD were more affected by muscle fatigue by the point at when the voluntary trials were completed at the end of the study.

The slower shoulder movement demonstrated by individuals with PD did not translate into a slower wrist movement or a delay in contacting the handrail. Thus, PD does not appear to lead to any functional consequences in producing perturbation-evoked or voluntary arm movements. Furthermore, the general lack of interactions and group

main effects observed in the current study may have been due to the testing of individuals with PD who were of low to mild disease severity and were still leading active lives (e.g., many reported participating in weekly exercise classes). Greater differences may have been observed if individuals who were more severely affected by PD were examined. The lack of group differences observed may have also resulted due to the relative simplicity of the voluntary reaching task or to the sensory information that remained available (i.e., visual input) during the voluntary arm movement condition. This is based on findings that have shown greater deficits in individuals with PD (i.e., less accuracy, lengthened transport time, and a decreased peak velocity of the wrist and trunk movement) during voluntary arm movements under more complex conditions, or when sensory information was manipulated (i.e., vision of the target object was removed, forcing an increased reliance on proprioceptive information about the hand) (Rand, et al., 2010). The absence of visual information has also previously been shown to result in less accurate voluntary arm movements in individuals with PD (Adamovich, Berkinblit, Hening, Sage, & Poizner, 2001; Keijsers, Admiraal, Cools, Bloem, & Gielen, 2005).

The finding of lower peak shoulder abduction angular velocities by individuals with PD exhibited seems to contradict the observation that of a larger relative MD EMG response in individuals with compared to without PD. However, there are some possible explanations for these apparently contradictory results. First, previous research has suggested that individuals with PD are capable of producing large bursts of EMG (Berardelli, et al., 1986); however they have difficulty matching the size of the muscle activity to the required movement (Berardelli, et al., 1986). As such, this could explain why the shoulder was moved slower in comparison to individuals without PD, despite the

large MD EMG activity. Second, it may be that along with activating the MD, perhaps individuals with PD also simultaneously activated muscles opposing shoulder abduction (e.g., the pectoralis major). Co-contraction of agonist and antagonist muscles has previously been reported in the trunk musculature of individuals with PD, in response to postural perturbations (Carpenter, et al., 2004; Dimitrova, et al., 2004). If co-contraction of the shoulder abductors and adductors occurred in this study, it could have influenced the speed at which individuals with PD were able to move their upper arm toward the handrail. Third, the method of normalizing MD EMG activity to each participant's MVC may be problematic if participants were unable to achieve a true maximal contraction. Specifically, if individuals with PD had difficulty in achieving a maximal contraction, their normalized percentages would have resulted in relatively larger MD EMG responses. Yet in absolute terms, the individuals with PD may have generated less muscle activity in response to each surface translation. In order for future studies to more accurately compare EMG data between groups, EMG data could be normalized to electrically-evoked maximal M-wave responses. The advantage to this method would be that a maximal M-wave is a better representation of a 100% contraction and would not be affected by a participant having difficulty in achieving a MVC after just completing an entire experimental protocol.

5.4 Limitations:

It is acknowledged that the current study was not without limitations. First, it cannot be determined whether the benefits of instruction observed in the current study would persist as disease severity progresses. This is because most individuals with PD

who participated in the current study exhibited a mild disease severity, with no participant exhibiting a high disease severity. Additionally, based on how well the individuals with PD performed on the questionnaires and functional assessments (i.e., ABC Scale, TUG, etc.) it does not appear that the participants with PD were representative of the more typical physically inactive individual living with PD.

With respect to the EMG collection, one limitation was that EMG recordings were only taken from one muscle of the upper arm (i.e., the MD). Therefore, it is not known whether the muscles of the forearm, which are involved in the actual grasping of the handrail, would positively benefit from explicit instruction.

While the kinematic outcome measures from the current study focused on quantifying the compensatory arm reaction and how it was influenced by instruction, the current study cannot determine whether the facilitated compensatory arm reactions actually resulted in an increase in mechanical stability. In the future, it would be interesting to measure variables such as COM movement, as this would be useful in determining whether the compensatory arm reactions elicited with explicit instruction were beneficial in preventing the COM from moving beyond the participant's BOS, compared to the react natural condition.

Lastly, the current study did not quantify participant gaze (e.g., through the use of an eye tracker). This information would confirm whether the benefits of explicit instruction were a result of a more frequent participant gaze at the handrail, allowing individuals to use visual input instead of the basal ganglia for producing compensatory arm reactions. Future studies may wish to incorporate eye tracking equipment to determine whether the use of explicit instruction to reach for and grasp a handrail

increases gaze fixation toward the handrail and if this is the case, when this fixation occurs (e.g., pre or post surface translation onset).

5.5 Conclusion:

First, explicit instruction to respond with a desired balance recovery strategy resulted in earlier and larger compensatory arm reactions in older adults with and without PD. No additional benefits were observed for the individuals with PD, as both groups benefitted from the instruction in a similar manner. Second, the comparison of the perturbation-evoked movements to the voluntary arm movements did not reveal either type of movement to be differentially affected by PD, suggesting that PD influences both types of arm abduction movement in a similar manner. With respect to the influence of instruction on compensatory arm reactions, the findings from the current study cannot establish whether the benefits of instruction would continue as the disease progresses, whether they persist if participants are re-tested at a later date and most importantly, whether they translate in response to a loss of balance in real life. If future studies could show that the benefits of instruction persist over time, rehabilitation professionals could employ explicit instruction to help train compensatory arm reactions in individuals with PD.

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